ARCHIVES OF OPHTHALMOLOGY

VOLUME 34 (old series Volume 91) OCTOBER 1945 NUMBER 4

COPYRIGHT, 1945, BY THE AMERICAN MEDICAL ASSOCIATION

EFFECT OF LOCAL ANESTHETICS ON CELL DIVISION AND MIGRATION FOLLOWING THERMAL BURNS OF CORNEA

GEORGE K. SMELSER, PH.D., AND V. OZANICS, M.S.

NEW YORK

It has long been recognized that the frequent topical application of anesthetics delays the repair of epithelial injuries of the cornea. This observation has been made clinically (Stallard [1]), and more rigorously controlled comparisons have been made on laboratory animals on which treated and untreated standardized corneal abrasions have been inflicted (Gundersen and Liebman,[2] Friedenwald and Buschke [3]). Such studies have lead to attempts to secure less harmful drugs which are yet effective anesthetics.

The search for an anesthetic agent which does not inhibit healing is obviously of practical value. It is of equal interest to determine whether inhibition of healing processes is proportional to the anesthetic properties and is, therefore, possibly due to the same factors which produce anesthesia. In such a case normal healing rate and anesthesia would be incompatible. It is the purpose of this paper to show that anesthesia and normal healing are compatible, to compare the effect of various anesthetics on cell division and migration of regenerating epithelium and to indicate some drugs which produce adequate surface anesthesia and yet inhibit healing very little.

There are two processes involved in the healing of epithelial wounds: (1) cell migration, to cover the injured area, and (2) mitosis, to reconstitute the normal number of epithelial cells. It has been generally accepted that the migratory activity occurs first and the cell division second. In the repair of small wounds, mitosis was found actually to decrease during the migratory phase (Arey,[4] Wigglesworth,[5] Arey and Covode,[6] Mann,[7] Friedenwald and Buschke [3]).

In earlier experiments (Smelser and Ozanics [8]) considerable mitotic activity was noted during the migratory phase of healing following rather extensive thermal burns of the cornea. In these experiments the number of mitotic figures found in the burned eye was greater than that found in the intact, control, eye in 84 per cent of the animals studied. These results were based on a study of thermal burns covering about one third of the cornea, whereas the earlier experiments of Arey, Arey and Covode, and Friedenwald and Buschke were concerned with small abrasions. In other experiments it was shown that abrasions, similar in size to the burns, healed in 64 per cent of the cases without significant increase in the number of mitoses.

Since cell division appears to play a role early in the healing of thermal burns, and since this type of injury was readily reproducible and produced no obvious damage to the substantia propria, it was decided to investigate the effect of anesthetic drugs on the healing of thermal burns.

Male rats of the Sherman strain, 80 to 105 Gm. in body weight and 5 to 6 weeks of age, were used. Comparisons of the effect of various drugs were drawn between litter mates. The horizontal band-shaped burns were produced with a Shahan thermophore equipped with a terminal fitted to the curvature of the rat's cornea. The animals were operated on while under

From the Department of Ophthalmology, Columbia University College of Physicians and Surgeons.

Read at a meeting of the New York Academy of Medicine, Section of Ophthalmology, Nov. 20, 1944.

The work described in this paper was done under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and Columbia University.

1. Stallard, H. B.: Methods of Anesthesia and Analgesia for Ophthalmic Operations, in Ridley, F., and Sorsby, A.: Modern Trends in Ophthalmology, London, Butterworth & Co., Ltd., 1940, chap. 49.

2. Gundersen, T., and Liebman, S. D.: Effect of Local Anesthetics on Regeneration of Corneal Epithelium, Arch. Ophth. **31**:29 (Jan.) 1944.

3. Friedenwald, J. S., and Buschke, W.: The Influence of Some Experimental Variables on the Epithelial Movements in the Healing of Corneal Wounds, J. Cell. & Comp. Physiol. **23**:95, 1944.

4. Arey, L. B.: Wound Healing, Physiol. Rev. **16**:327, 1936.

5. Wigglesworth, V. B.: Wound Healing in an Insect (Rhodnius Prolixus Hemiptera), J. Exper. Biol. **14**:364, 1937.

6. Arey, L. B., and Covode, W. M.: The Method of Repair in Epithelial Wounds of the Cornea, Anat. Rec. **86**:75, 1943.

7. Mann, I.: Study of Epithelial Regeneration in Living Eye, Brit. J. Ophth. **28**:26, 1944.

8. Smelser, G. K., and Ozanics, V.: Effect of Chemotherapeutic Agents on Cell Division and Healing of Corneal Burns and Abrasions in the Rat, Am. J. Ophth. **27**:1063, 1944.

ether anesthesia, with the same technic as that described earlier.[8] The thermophore (71 C.) was applied for five seconds to both eyes, and autopsy was performed twelve hours later. The material to be tested was placed on the right eye every two hours during this period, and the rat was held so that the solution or ointment remained on the cornea and conjunctiva for one minute. Thus the epithelium was in contact with the drug for a minimum of five minutes during the twelve hour period. After treatment the rats quickly rid themselves of the ointment or solution. The eyes were fixed in Bouin's fluid, and the outline of the burned area was drawn with a camera lucida at a magnification of 30 diameters. Untreated burns, of the size studied, became covered with epithelium in fourteen hours and were nearly healed in twelve hours. Camera lucida drawings were made of the treated and the control burns. The edge of the regenerating epithelium was clearly visible in the eyes fixed in Bouin's fluid. Since the wounds were nearly healed, the area drawn was almost in a plane; however, when the injured area was still large, the area represented in the drawing was a little less, owing to the projection of a curved surface on to a plane one. This resulted in a conservative estimate of delays in healing. The area of the unhealed portion of the burn was determined by planimetric measurements of the drawings. The eyes were then embedded and sectioned, and the number of mitotic figures in the regenerating epithelium was counted. Sufficient counts were made so that repeatable values were obtained. The technic and the statistical methods used were the same as those reported earlier. The effect of the treatment given was judged in each case by comparing the mitotic figure counts and the residual area of the burn of the treated and the control eye of the same animal. These burns were found to be reproducible. In a series of 10 rats both eyes were burned and no treatment was applied. Both eyes healed at essentially the same rate, and the number of mitotic figures found in the right and the left eye was not significantly different in any case. The average area of the burn in the right eye of these rats varied only 7 per cent from the average area of the burn in the left eye. Variation between individual rats, however, was much greater.

EFFECT OF ANESTHETICS ON CELL DIVISION IN THE INTACT EPITHELIUM

As a preliminary to the experiments on regenerating epithelium, the effect of several anesthetics on mitosis in the intact corneal epithelium was determined. No apparent damage of the cornea was produced by these experiments, such as was described by Baba,[9] who used the drugs in much greater concentrations. Solutions of cocaine hydrochloride, 0.5 per cent; tetracaine (pontocaine) hydrochloride, 1 and 0.5 per cent; nupercaine hydrochloride, 0.5 per cent; phenacaine (holocaine) hydrochloride, 2 and 0.5 per cent; butacaine sulfate, 2 per cent, and chlorobutanol, 1 per cent, and ointments containing butyl aminobenzoate (butesin), 0.75 per cent; orthoform, 2 per cent, and butacaine (butyn) sulfate, 2 per cent, and metaphen 1:3,000 were used.[9a] The aqueous solutions were unbuffered and contained 1 per cent sodium chloride, except the preparation of butacaine sulfate, which was made up in distilled water. The *p*H of the solutions, determined with a glass electrode, ranged from 4.0 to 6.0. These preparations were placed on one eye for one minute each hour for eight hours. The eyes were fixed and sectioned, and the number of mitotic figures in the epithelium was counted in each eye. The ratio of the number found in the treated to that in the untreated, control, eye was expressed as a per cent and the significance of the difference calculated.

The results of these experiments are shown in a bar graph (fig. 1). Application of all but four of these compounds inhibited cell division when concentrations which are useful clinically were employed. Whereas the average depression of mitosis caused by 0.5 per cent cocaine hydrochloride and 1 per cent tetracaine hydrochloride

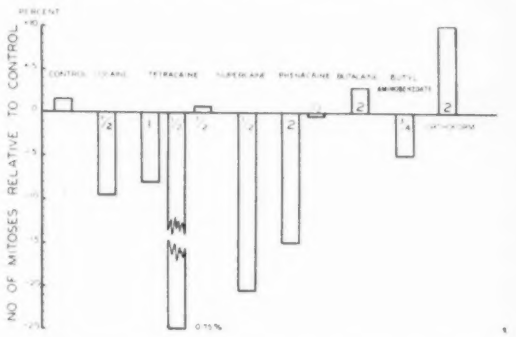


Fig. 1.—Effect of anesthetics on mitosis in the intact corneal epithelium. Each bar represents the average of 4 to 7 eyes; the control, the average for 10 eyes. The figures in the bars represent the concentration of the drug expressed as a per cent.

was slight, both 0.5 per cent nupercaine hydrochloride and 2 per cent phenacaine hydrochloride caused a rather marked decrease in the number of mitotic figures found in the treated corneas. More dilute solutions of tetracaine and phenacaine had no deleterious effect on cell divi-

9. Baba, H.: Vergleichende Beobachtungen über die durch Instillationen von verschiedenen lokalen Anaesthetica hervorgerufenen Augenveränderungen, abstracted, Zentralbl. f. d. ges. Ophth. **40**:555, 1938.

9a. The solutions of cocaine hydrochloride were obtained in the hospital pharmacy. The solutions of all other drugs were prepared in our laboratory. Some of the ointments were also prepared in our laboratory in the hydrous wool fat base mentioned; others were proprietary ointments, obtained as follows:

Ophthalmic Ointment Butyn Sulfate 2% and Metaphen 1:3,000, Abbott Laboratories; Ophthalmic Ointment Butesin Picrate 1% and Butesin 1%, Abbott Laboratories; Pontocaine Base Eye Ointment (0.5 per cent tetracaine base), Winthrop Chemical Company, Inc.; Nupercainal (nupercaine base, 1 per cent), Ciba Pharmaceutical Products, Inc., and Holocaine Hydrochloride (2 per cent phenacaine hydrochloride), Abbott Laboratories. These products were purchased locally.

sion in the cornea. Butacaine sulfate 2 per cent, was the only drug tested which could be applied in so great a concentration without causing a decrease in the number of mitotic figures. Applications of an ointment containing 2 per cent butacaine sulfate likewise did not affect cell division. Two fat-soluble anesthetics were studied, butyl aminobenzoate and orthoform. These compounds were dissolved in a hydrous wool fat base (anhydrous wool fat, 75 per cent; water 25 per cent) in concentrations of 0.75 and 2 per cent, respectively. Neither preparation markedly affected cell division. The data obtained for the orthoform group were somewhat variable; in 2 of the 5 tests definitely more mitoses were found in the treated cornea, thus raising the average response in this group. The 6 animals treated with chlorobutanol also gave a somewhat surprising and variable response. Four of the 6 corneas treated with chlorobutanol had more mitotic figures in the epithelium than the untreated, control, cornea. One animal showed a definite depression.

These experiments show that most of the anesthetics tested affected mitosis adversely. This effect is, of course, modified by the concentration of the drug used, the route of administration and the frequency of application. This last factor was tested with tetracaine. Instead of the application of 0.5 per cent solutions once an hour, which was shown to have no effect on cell division, three applications an hour were made. The technic was not otherwise varied. The frequent, in contrast to the hourly, application of tetracaine was found to cause an extreme and consistent depression in cell division (75 per cent fewer figures than in the control eye).

The effect of subconjunctival injections of anesthetics on corneal mitosis was studied with cocaine hydrochloride, 1 per cent, and procaine hydrochloride, 2 per cent, in 8 animals. This type of experiment was difficult to evaluate because the volume of solution injected (0.05 cc.) was so great relative to the volume of the globe and orbit that some displacement of the soft tissues was probably produced. The results, therefore, are not comparable to those of subconjunctival injections in man. The corneas were fixed for study five to seven hours after the injections. At this time there was a marked depression in the mitotic figures seen in the eye in which cocaine had been injected. The effect of procaine, as would be expected, was less pronounced, though consistent; the number of figures found averaged 20 per cent less than in the control eye. When 2 per cent procaine hydrochloride was made up in a solution of epinephrine hydrochloride (1:2,500) no mitotic figures at all were found in the corneal epithelium of the eye in which the anesthetic was injected. This result may have been due in part to a prolongation of the effect of procaine, but undoubtedly the epinephrine itself inhibited cell division (Friedenwald and Buschke [10]).

EFFECT OF LOCAL ANESTHETICS ON MITOSIS IN REGENERATING EPITHELIUM

In general, anesthetics had a more pronounced depressing action on mitosis following burns than in the intact epithelium (fig. 2), even though the drugs were applied once in two hours instead of every hour, and in some cases in lower concentrations. Two exceptions to this rule, phenacaine and butyl aminobenzoate, were noted. Both of these drugs interfered less with cell division in regenerating than in normal epithelium. When the more dilute preparations, 0.5 and 0.75 per

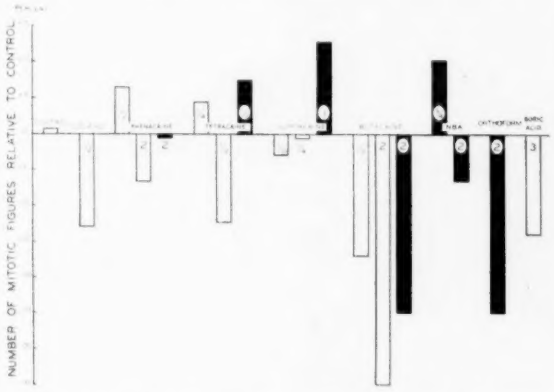


Fig. 2.—Effect of anesthetics on mitosis in the corneal epithelium twelve hours following a thermal burn. Each bar represents the average for 5 to 7 eyes. The right eye of each animal was treated with the anesthetic; the control is the other, untreated but burned, eye of the same animal. The figures in the bars represent the concentration of the drug expressed as a per cent. *N B A* indicates normal butylaminobenzoate.

In this figure the hollow bars represent aqueous solutions; the black bars, ointments.

cent, were applied, more mitoses were seen than in the untreated control burns of the other eye; however, the increase could not be considered statistically significant in either case.

The anesthetics may be arranged in order of their inhibitory action on cell division in the regenerating epithelium. Butacaine sulfate was the worst in this respect (fig. 2). Since aqueous solutions of butacaine sulfate also retarded

10. Friedenwald, J. S., and Buschke, W.: The Effects of Excitement, of Epinephrine and of Sympathectomy on the Mitotic Activity of the Corneal Epithelium in Rats, Am. J. Physiol. **141**:689, 1944.

epithelization, the resulting smaller epithelial area may of itself have led to somewhat lower counts. Cocaine and tetracaine, which were next, and essentially equal, inhibited mitosis less. Phenacaine, butyl aminobenzoate and nupercaine were the best in that their application did not greatly affect the number of mitotic figures present in the healing burn. It is notable that the effect of butacaine and orthoform was very different on the injured and on the intact epithelium. The difference in the effect of nupercaine on the intact and the burned epithelium was probably due to the lower concentration used.

The superiority of ointments over aqueous solutions of several drugs was particularly noticeable in their effect on mitosis. Ointments containing 2 per cent butacaine sulfate (two preparations) inhibited cell division less than 2 per cent aqueous solution applied with the same frequency. Phenacaine, tetracaine and nupercaine ointments had no deleterious effect on

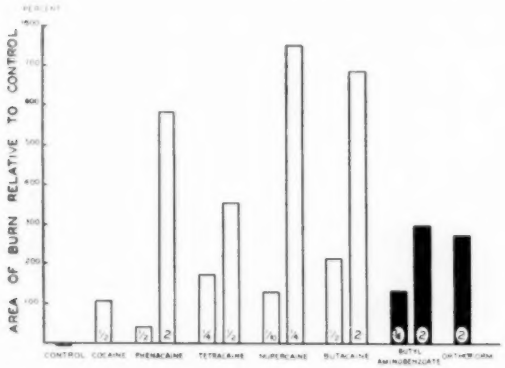


Fig. 3.—Influence of anesthetics on the healing of corneal burns. Each bar represents the average area of treated corneal burns relative to the average area of the untreated burns in the opposite corneas of the same animals. The measurements were made twelve hours after injury with a planimeter from camera lucida drawings. Five to 10 animals are represented by each bar. The hollow bars represent aqueous solutions; the black bars, ointments.

mitosis, whereas aqueous solutions of these drugs reduced the number of mitotic figures found. The nupercaine and tetracaine ointments contained the drug as its base rather than as the salt, which was used in the other cases.

EFFECT OF LOCAL ANESTHETICS ON CELL MIGRATION IN A REGENERATING EPITHELIUM

The second phase of healing, i. e., migration of the epithelial cells to cover the denuded stroma, was also notably inhibited by some of the anesthetics. This observation is in agreement with those of Friedenwald and Buschke [11] on the effect of some anesthetics on cell movement in vitro. All of the compounds studied slowed this process but differed in the degree of the effect produced (fig. 3). It appeared that migration was more easily inhibited than mitosis, although an exception to this rule (butacaine ointment) was found. The effect of each drug was highly consistent, since an inhibition was always produced, although it was variable in its severity. Just as the drugs studied could be rated in their effect on mitosis, a similar tabulation was possible with respect to their influence on cell migration. Aqueous solutions of nupercaine had, of all of the drugs tested, the greatest inhibitory action on cell movement. A 0.1 per cent solution of nupercaine hydrochloride was approximately equal to a 0.5 per cent solution of cocaine hydrochloride in this respect. In the table, the drugs are listed in descending order of their adverse effect on this healing process. Factors other than the active drug itself, such as the p_H or the tonicity of the solutions, may have been responsible for some of the inhibitory action. A solution of boric acid, p_H 4.1, inhibited cell migration, but to a slight extent as compared with the anesthetics.

The two fat-soluble drugs, orthoform and butyl aminobenzoate, were dissolved in hydrous wool fat and applied as ointments. In 2 per cent concentration they both inhibited cell migration rather markedly, though less than the other drugs in aqueous solutions of similar concentrations (fig. 3). Control tests with the same hydrous wool fat base also hindered cell migration but no more than did a solution of boric acid. A mixture of 1 per cent butyl aminobenzoate and 1 per cent butesin picrate made up in petrolatum was much less detrimental to cell migration than the butyl aminobenzoate alone in hydrous wool fat.

In addition to the experiments with solutions, four of the drugs, nupercaine, butacaine, tetracaine and phenacaine, were also applied as ointments. A comparison of the results of the application of anesthetics in these two forms on cell migration is shown in figure 4. The ointment base varied in the four preparations, and two of the drugs (nupercaine and tetracaine) were in the form of their bases, whereas phenacaine and butacaine were salts. In all four series the ointments had a far less deleterious effect than did the solutions. Ointments of nupercaine base, butacaine sulfate and phenacaine hydrochloride

11. Friedenwald, J. S., and Buschke, W.: Mitotic and Wound-Healing Activities of Corneal Epithelium, Arch. Ophth. **32**:410 (Nov.) 1944.

did not inhibit healing more than a hydrous wool fat ointment, whereas the aqueous solutions of these compounds greatly retarded this process. Indeed, the injuries treated with solutions of nupercaine hydrochloride and butacaine sulfate had remained at nearly their initial size for twelve hours, while untreated control burns had almost completed epithelization. Such an enormous difference between the solutions and the ointment was not found with tetracaine; however, this drug was used in lower concentration (0.5 in concentration, the superiority of the ointment over the solution is obvious. A 1 per cent solution of nupercaine hydrochloride is too great a concentration to use on the injured rat eye.

It is obvious from the foregoing observations that the drugs tested differed in their effect on the two processes studied. In general, the ointments were preferable to the solutions, and of these, phenacaine and nupercaine ointments had the least effect on either process. If mitosis is disregarded, butacaine ointment ranks with these

Relative Effect of Anesthetics on Mitosis in the Intact, and Cell Division and Migration in the Burned, Corneal Epithelium *

	Most Inhibitory		Least Inhibitory
		Aqueous Solutions	
Intact			
Mitosis	Nupercaine;	cocaine; phenacaine = tetracaine;	butacaine
Burned			
Mitosis	Butacaine;	cocaine = tetracaine; nupercaine, 0.25%;	phenacaine
Migration	Nupercaine;	tetracaine; butacaine; cocaine;	phenacaine
		Ointments	
Intact			
Mitosis	Cocaine, 0.5%;	butyl aminobenzoate, 0.75%; butacaine sulfate, 2%;	orthoform, 2%
Burned			
Mitosis	Orthoform, 2% = butacaine sulfate, 2%;	butyl aminobenzoate, 2%;	phenacaine hydrochloride, 2% = tetracaine base, 0.5% = nupercaine base, 1%
Migration	Orthoform, 2% = butyl aminobenzoate, 2%;	tetracaine base, 0.5%; nupercaine base, 1%;	phenacaine hydrochloride, 2% = butacaine sulfate, 2%

* The order in which the drugs are listed should not be considered as an indication of their absolute position in the series, for many were approximately equal in their effect (indicated by =). The comparisons were made between equal per cent concentrations except where noted.

per cent) than were most of the others. It will be noted that an ointment containing 1 per cent nupercaine base was compared with 0.25 per cent solution of the salt. Even with this difference in the slight effect on migration; tetracaine ointment, on the other hand, did not interfere with mitosis but delayed cell migration more than did the others.[11a]

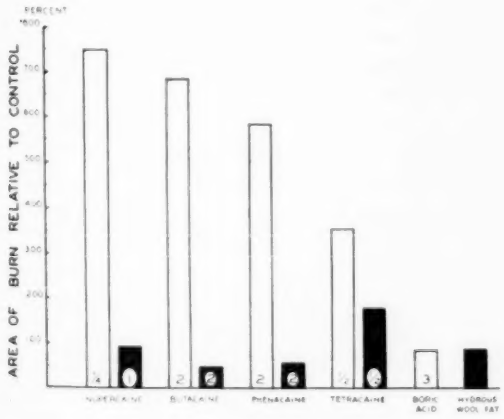


Fig. 4.—Comparison of the inhibitory effect of anesthetic ointments and solutions on epithelization of corneal burns. Five experiments are represented by each bar. (Some of the data are the same as those shown in figure 3.) All of the data were obtained as described in the legend of figure 3. The hollow bars represent aqueous solutions; the black bars, ointments.

COMMENT

The experiments just described confirm the well established opinion that many anesthetics interfere with healing. They show, further, that the drugs commonly used vary in their effect on healing, as they also do in their ability to pro-

11a. Since this article was submitted for publication, we have carried out similar experiments with metycaine hydrochloride (Lilly) solutions and ointments. The burned areas, treated with a 1 per cent solution of metycaine hydrochloride, were 134 per cent larger than the untreated control burns. This delay in healing was similar to that caused by a 0.1 per cent solution of nupercaine hydrochloride. There were 17 per cent fewer mitotic figures in these treated eyes, a depression similar to that caused by butacaine sulfate. The ointment containing 4 per cent metycaine hydrochloride delayed epithelization a little less than did the ointment containing 0.5 per cent tetracaine base. The burned area was 159 per cent greater than the average area of the control burns. The number of mitotic figures was 25 per cent less in the ointment-treated eyes, an effect similar to that obtained with the ointment containing butacaine sulfate, 2 per cent, and metaphen.

duce anesthesia. It has been possible to classify the several drugs approximately in the order of their inhibitory effect on these processes (table). In addition, it appears that an anesthetic does not necessarily affect mitosis and cell migration equally.

Although cell division and cell migration both involve the movement or flowing of protoplasm and the biologic basis for both processes may be fundamentally the same, the two processes can be inhibited separately, and cell migration appears to be much more easily inhibited by some drugs than cell division. For example, aqueous solutions of phenacaine and nupercaine markedly inhibited cell migration but had no appreciable effect on the number of mitotic figures found in the epithelium of these corneas. The reverse condition was found to obtain when a butacaine and metaphen ointment was employed. An explanation of the basic mechanism involved would undoubtedly take account of the action of the drugs on the intracellular physiology of these processes, as shown in the studies of Michaelis and Quastel,[12] and of Herrmann, Moses and Friedenwald[13] on narcotics and respiratory enzymes. A second possibility which should not be ignored is suggested by the fact that some of the drugs used (nupercaine) were rather good detergents. It may be that this property is responsible in part for the inhibition of epithelization. Strong detergents would act in this manner; furthermore, when the drugs were incorporated in ointments, in which the detergent action would be reduced, they failed to delay significantly cell migration.

Although for the purposes of the experiment the drugs were applied frequently, once every two hours, it is worth while to note that the complete anesthetization of the cornea with several applications of cocaine produced an effect which was readily discernible under the conditions of our experiments many hours later, presumably long after the anesthesia had worn off. Comparison of the effects of various anesthetics, made on the basis of the molar rather than the per cent concentration of the drugs, would provide a more exact basis on which to rate the different compounds. However, the drugs used, except butyl aminobenzoate and orthoform, were fairly similar in molecular weight. 1 per cent solutions varying from one-thirty-fifth to one-thirty-ninth molar. Therefore, in view of the nature of the experiments, satisfactory comparisons may be made on a percentage concentration basis.

The fact that more mitotic figures were found in the eyes treated with some of the drugs (fig. 2) was not interpreted as due to stimulation of mitosis. The data were variable and the differences not statistically significant.

A more interesting and practical basis for comparison would be to relate the inhibition of healing to the degree of anesthesia produced. Determination of the anesthetic properties of the several drugs lies outside the province of this study; however, tests were made on the intact human cornea in which the duration of anesthesia produced by these drugs under standard conditions was compared. Although the tests were preliminary in nature, they showed that the more dilute aqueous solutions used on the rats produced corneal anesthesia of short duration. Anesthesia following a one minute contact with a 0.5 per cent solution of phenacaine hydrochloride or butacaine sulfate persisted fifteen to twenty minutes, and that produced by 0.1 per cent nupercaine hydrochloride or 0.25 per cent tetracaine hydrochloride lasted twenty-five to thirty minutes. There was not a good correlation, therefore, between duration of anesthesia and inhibition of cell migration, for butacaine produced much delay in healing with a short period of anesthesia, whereas tetracaine gave a longer period of anesthesia with about the same delay in cell migration. However, the data on duration of anesthesia are few and were obtained from another species and on an intact rather than a broken epithelium.

The experiments with anesthetics in an ointment base clearly demonstrated that duration of anesthesia had no relation to the inhibition of healing. Figures 2 and 4 show that ointments containing an anesthetic had a far less deleterious effect on cell division and cell migration in the burned cornea than did aqueous solutions of the same drugs. These ointments also produced anesthesia which persisted far longer (one and a half to two hours) than did that produced by the aqueous solutions. It seems probable therefore (always assuming a similarity of anesthetic effect between rat and man) that the burned eyes were under anesthesia during the entire period of wound healing after the first application of the ointment without any appreciable slowing down of cell migration and, except for butacaine, without inhibition of mitosis. The fat-soluble anesthetics butyl aminobenzoate and orthoform in ointments both inhibited cell division and migration; the latter effect was greater with orthoform, whereas butyl aminobenzoate

12. Michaelis, M., and Quastel, J. H.: The Site of Action of Narcotics in Respiratory Processes, Biochem. J. **35**:518, 1941.

13. Herrmann, H.; Moses, S. G., and Friedenwald, J. S.: Influence of Pontocaine Hydrochloride and Chlorobutanol on Respiration and Glycolysis of Cornea, Arch. Ophth. **28**:652 (Oct.) 1942.

produced anesthesia of greater duration. From these experiments it seems clear that anesthesia is compatible with an almost normal rate of healing both in the cell division and in the cell migration phase. The reason for the superiority of ointments over aqueous solutions in this respect is not clear; however, the epithelial cells bathed in an aqueous solution of an anesthetic endure a very high concentration of the drug for a rather short time. The ointments may be presumed to release their drug rather slowly over a more extended period, so that the concentration of the anesthetic may not reach toxic levels at any time.

SUMMARY OF RESULTS

1. Topical application of most anesthetics inhibits mitosis in the intact corneal epithelium when concentrations which produce anesthesia are used.

2. The inhibition of cell division varies with different drugs, the method of administration and the frequency of application.

3. Anesthetics applied locally to corneal burns markedly inhibit the migration of epithelial cells over the injured area.

4. Cell division in the regenerating epithelium is also inhibited by most anesthetics.

5. The processes of cell migration and cell division are affected differentially. Cell migration may be notably retarded with little or no inhibition of cell division. Other drugs inhibit mitosis but do not markedly retard cell migration.

6. Some anesthetics (nupercaine and phenacaine ointments) can be used which produce prolonged surface anesthesia and which do not impair mitosis in the normal, or retard cell division or migration in regenerating, corneal epithelium.

7. Unbuffered aqueous solutions inhibit the healing processes far more than ointments of the same or greater strength, which produce anesthesia of longer duration.

630 West One Hundred and Sixty-Eighth Street.

STANDARD ILLUMINANTS IN RELATION TO COLOR-TESTING PROCEDURES

LE GRAND H. HARDY, M.D.
NEW YORK

THE PROBLEM

What is wrong with the present color-testing procedures? Why do examiners often fail to expose persons with defective color vision? Does the fault lie in the tests, the methods of administering them or the conditions under which they are administered?

IMPORTANCE OF THE PROBLEM

In the Army.—An officer is shot because a cadet pilot fails to recognize a colored signal and fires an extra round. A bomber is shot down because a returning pilot fails to respond to a prearranged color signal code. Data of the Civil Aeronautics Administration show that a pilot who failed to pass the Ishihara test on the first attempt has a 50 per cent chance of passing a retest.

In the Navy.—Tests conducted by members of the Committee on Color Blindness of the Inter-Society Color Council show that all the commonly used tests for color blindness are unreliable and largely ineffective as they are ordinarily administered.

In Industry.—Because the wrong illuminant was used in making color matches, $50,000 worth of silk is ruined. Because of faulty judgment of color, 100,000 pounds (50,000 Kg.) of cotton is graded down.

A sheet metal printer in one instance lost the time and material for more than 780,000 talcum cans on which the color went wrong because some of the work was done under ordinary artificial light.

A lithographer was refused acceptance on a million calendars with a trademark design because the color, which appeared similar to the approved copy sheet under artificial light, was quite different and unsatisfactory in daylight. These calendars were worth only their weight in scrap paper.[1]

Physicians, especially ophthalmologists, should be interested in this problem, for it is their function to carry out these tests for color blindness or defective color vision. Yet few ophthalmologists have any adequate understanding of the principles underlying tests for color vision or of safeguards essential to their administration. This is due partly to lack of training and to the fact that defective color vision is an anomaly and not a clinical disease.

Any test of color vision involves three equally important factors: (1) the illuminant to be used; (2) the subject to be tested, and (3) the test and its proper administration.

In such a situation any one of the factors may be tested if, and only if, the other two are standardized or constant. Yet it is the rule rather than the exception to leave two of the factors (1 and 2) variable and thus hopelessly muddle any results obtained. The light (factor 1) is an instrument to be used in order to enable the subject (factor 2) to perform a certain task (factor 3). It is obvious that if on one occasion the subject is given a wretched instrument and on another occasion he is given a perfect instrument, he will perform differently on the two tests. At county fairs one tests one's strength by striking a blow on one end of a lever, the other end of which drives a ball up a guide wire. Other things being equal, it is obviously no test of strength if one man is given a tack hammer and the next man a sledge hammer. Considering the fact that color vision tests have been given to hundreds of millions of subjects, a fantastic amount of time and effort has been wasted through ignorance or disregard of this simple fact. It is established that by far the greater amount of work on color vision testing has been futile and invalid. Dimmick[2] has recently pointed out again that tests involving reflected light are "dependent for precision on the illuminant used."

Color vision tests are deliberately designed to be tricky and critical. The more critical they are the more accurate they are, but the more carefully must they be administered, and this

1. Gage, H. P., and Macbeth, N.: Filters for Artificial Daylighting: Their Grading and Use, Tr. Illum. Engin. Soc. **31**:995, 1936.

2. Dimmick, F. L.: Methodology in Test Preparation, Am. J. Optom. **20**:430, 1943.

means that more care must be used in selecting the illuminant used and in administering the test.

Most color vision tests as presently administered are not tests for defective color vision but tests for lighting systems. It is easier to test factor 1 (the illuminant) than to test factor 2 (the subject), and this is what is commonly done. Excellent tests, using metameric colors, are now available for testing illuminants, but ordinary tests intended to evaluate color vision are usually—although unwittingly and erroneously—so administered that they test primarily the illuminant.

If some one is seen to place something on the pan of a spring scale and observe the position of the indicator, what is the inference? The answer depends on a fuller knowledge of the purpose and conditions of the procedure. If it is the first session of a high school physics class, one may be seeing tested the ability of the student to read a vernier; if it is in a grocery store, one is probably seeing a mass of spinach being weighed; however, if the scene takes place at the Bureau of Standards one is more likely seeing a set of scales being tested. But in testing color vision one always, unfortunately, assumes that it is spinach.

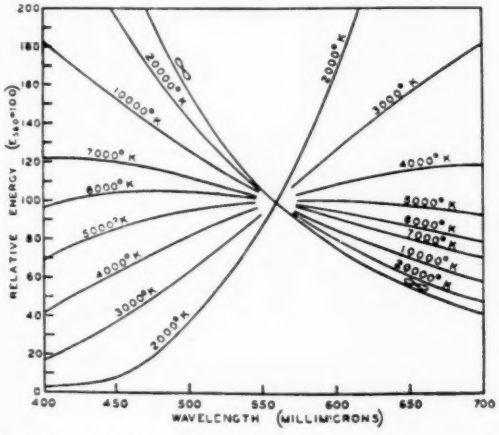


Fig. 1.—Relative distribution of energy in the radiation from a black body at various temperatures.

Even the pseudoscientific who always use "daylight" fall into this error, for their "10 foot candle daylight," as will be shown, varies from candlelight to light much bluer than average north skylight. The stage has been passed where anything which comes in through a window can scientifically be called "daylight illumination." Properly administered, practically all color tests using reflected light are independent of the amount of illumination between 10 and 60 foot candles.

BLACK BODIES AND APPARENT COLOR TEMPERATURE

Incandescent gases radiate line spectrums. In solids the atoms are so closely packed that they cannot emit radiations in a characteristic manner; as a result, a continuous spectrum is emitted. The emission is practically independent of the composition of the solid but depends primarily on its temperature. A black body is the perfect radiator. Such a radiator cannot

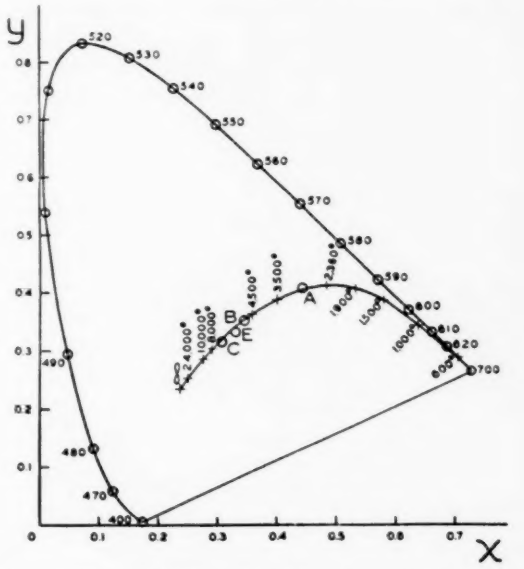


Fig. 2.—Chromaticities of various important illuminants. The solid line is the locus of radiation from a black body at various temperatures. The points *A*, *B* and *C* represent the chromaticity of the I. C. I. illuminants. The point *E* represents the chromaticity of a source radiating equal amounts of energy in each wavelength interval.

be achieved in actual practice, but a small hole in a large sphere whose walls are maintained at a constant temperature furnishes a close approximation.

If such a body is heated, it will emit infra-red radiations; and as the temperature is increased, it will become visibly "red hot." With further increases in temperature it becomes "yellow hot," then "white hot" and finally, theoretically, "blue hot." "Blue hotness" cannot be achieved experimentally because all known materials evaporate long before this temperature is reached.

However, measuring the distribution of the energy radiated by a black body at various wavelengths and different temperatures gives a series of curves which can be plotted as distribution curves on simple graph paper (ordinates represent relative energy emitted; abscissas, wavelengths) or reduced to points on a color diagram.

Such scales are shown in figures 1 and 2, taken from Hardy,[3] and figure 3, from Macbeth.[4]

From figure 3 it is seen that the sun seventeen minutes after sunrise or before sunset has the color temperature of a candle (1,900° K.). A tungsten vacuum type lamp has a color temperature of about 2,400° K.; a 100 watt, gas-filled Mazda C lamp, a color temperature of about 2,700° K., and a 200 watt Mazda C lamp, a color temperature of about 2,850° K. Noon sunlight at Washington, D. C., is only slightly yellowish, and its color temperature is a little above

Fig. 3.—Sunlight, daylight and artificial light on the color temperature scale (from Macbeth,[4] page 302). Data marked by ** indicate direct sunlight, time after sunrise and before sunset. In this chart, data marked by the * and ** were furnished by Priest, and those marked with a rectangle, by Cady and Dates; the data marked with an inverted triangle were obtained with a light source at 2898° K.

5,000° K. Uniform overcast skylight (6,000° to 7,000° K.) is an important illuminant, and the color temperature of blue northwest clear skylight may run up to 25,000° K. Hence "daylight" as expressing "what comes in through the window" has no significance and is largely pseudoscientific self delusion.

THE I. C. I. STANDARD ILLUMINANTS

There have been for the past thirteen years a set of international standard illuminants precisely specified and capable of accurate reproduction. These illuminants were accepted and recommended for any work in color by the International Commission on Illumination; hence they are known as the I. C. I. illuminants, sometimes called C. I. E. (*Commission Internationale de l'Éclairage*) illuminants. Sufficient time has elapsed and sufficient experimental and theoretic data have been produced to guarantee that the recommendations made in 1931 will serve for many years to come.

There are three recommended I. C. I. illuminants. They are called I. C. I. standard illuminants A, B and C. The official report of the

3. Hardy, A. C.: Handbook of Colorimetry, Cambridge, Mass., Massachusetts Institute of Technology, 1936.

4. Macbeth, N.: Color Temperature Classification of Natural and Artificial Illuminants, Tr. Illum. Engin. Soc., **23**:302, 1928.

recommendations was published by Smith and Guild [5] and was summarized for American readers by Judd [6] in the *Journal of the Optical Society of America,* from which the following quotation is taken.

It is recommended that the following three illuminants be adopted as standards for the general colorimetry of materials:

A. A gas-filled lamp of color temperature 2848°K.

B. The same lamp used in combination with a filter composed of a layer one centimeter thick of each of two solutions B_1 and B_2, contained in a double cell made of colorless optical glass. The two solutions are made up as follows:

Solution B_1

Copper sulphate ($CuSo_4.5H_2O$)	2.452	g
Mannite ($C_6H_8(OH)_6$)	2.452	g
Pyridine (C_5H_5N)	30.0	cc
Distilled water to make	1000	cc

Solution B_2

Cobalt ammonium sulphate ($CoSO_4.(NH_4)_2SO_4.6H_2O$)	21.71	g
Copper sulphate ($CuSO_4.5H_2O$)	16.11	g
Sulphuric acid (density 1.835)	10.0	cc
Distilled water to make	1000	cc

C. The same lamp used in combination with a filter consisting of a layer one centimeter thick of each of two solutions, C_1 and C_2, contained in a double cell made of colorless optical glass. The two solutions are made up as follows:

Solution C_1

Copper sulphate ($CuSO_4.5H_2O$)	3.412	g
Mannite ($C_6H_8(OH)_6$)	3.412	g
Pyridine (C_5H_5N)	30.0	cc
Distilled water to make	1000	cc

Solution C_2

Cobalt ammonium sulphate ($CoSO_4.(NH_4)_2SO_4.6H_2O$)	30.580	g
Copper sulphate ($CuSO_4.5H_2O$)	22.520	g
Sulphuric acid (density 1.835)	10.0	cc
Distilled water to make	1000	cc

It is recognized that for certain special applications (for example, the specification of signal glasses) other luminous sources may be prescribed, but in the absence of special conditions one of the three indicated sources should be used.

The liquid filter specified under illuminant *B* is the Davis-Gibson filter for converting color temperature 2848 to 4800°K, and that specified under illuminant *C* is the Davis-Gibson filter for converting 2848 to 6500°K. Illuminant *A* is intended to be typical of light from the gas-filled incandescent lamp, illuminant *B* is an approximate representation of noon sunlight, and illuminant *C* is an approximate representation of average daylight.

It is to be noted that I. C. I. illuminant A alone can be achieved from an unfiltered light source. Suitable I. C. I. A lamps that have been seasoned and calibrated may be obtained from the Bureau of Standards of the United States Department of Commerce and elsewhere. I. C. I. illuminant A can be taken as a standard for indoor Mazda lighting.

I. C. I. illuminant B has the same source as I. C. I. illuminant A, but the spectral energy distribution is modified by a filter. The exact specification requires two liquid filters, but these are necessary only for precise work. Glass filters are available which may be used in most practical circumstances to give a good approximation to I. C. I. illuminant B, which can be considered a standard for white light or sunlight.

Similarly close approximations to I. C. I. illuminant C, which may be considered as standard daylight, can be achieved by means of lamp and glass filter combinations,[7] which have the apparent color temperature of a black body at about 6,700° K. Unless otherwise specified, this is the illuminant that should be used in all color testing involving the use of yarns, plates, chips, etc.

Practically all color vision tests involving the use of reflected light were designed to be used under illumination approximating that given by I. C. I. illuminant C, and to employ them under other conditions not only invalidates the test but yields misleading results. The use of Mazda light in administering such tests as the Ishihara, the Stilling, the American Optical Company and the Rabkin plates and the plates prepared at the Knapp Memorial Laboratories of the Institute of Ophthalmology not only fails to trap but actually assists the escape from detection, particularly of deuteranomalous and mildly protanomalous subjects. Similar results have been found by the Navy Department.[8] It were better not to give the test than to give it under conditions which almost inevitably mislead.

SUMMARY

Tests for defective color vision always involve three factors: the light used to see with, the subject tested and the materials and procedures of the test. Failure to standardize two of the three factors causes invalid results in evaluating the third factor. Most color vision testing is carried out under conditions which make the procedure

5. Smith, T., and Guild, J.: The C. I. E. Colorimetric Standards and Their Use, Tr. Am. Optic. Soc. **33**:5, 1931-1932.

6. Judd, D. B.: The 1931 I. C. I. Standard Observer and Coordinate System for Colorimetry, J. Optic. Soc. America **23**:359, 1933.

7. The filters may be obtained from Corning Glass Works; the lamp and filter combinations, from the Macbeth Daylighting Company.

8. Reed, J. D.: The Effect of Illumination in Changing the Stimuli in Pseudo-Isochromatic Plates, J. Optic. Soc. America **34**:350, 1944.

as much a test of the illumination as of the subject's color discriminatory ability.

Standard illuminants for testing are available and must be used if reproducible results are to be obtained.

The illuminant must be an integral part of every accurate test.

I. C. I. illuminant C is the choice for all work in color vision testing which involves the use of reflected light and, unless special conditions are indicated, should always be used. A sufficiently close approximation to this illuminant is commercially available at relatively low cost.

Attention to this factor will enormously reduce the errors and misinterpretations now so prevalent in color vision testing.

23 East Seventy-Ninth Street.

SENILE HYALINE SCLERAL PLAQUES

KENNETH L. ROPER, M.D.
CHICAGO

The recent literature describes two new lesions of the sclera—one primarily necrotic in origin and the other primarily degenerative in character.

Within a period of six months I have observed 5 cases of the latter type. The condition is one of symmetric hyaline degeneration of the sclera, occurring in advanced age and characterized by the presence of slate gray plaques, situated about 3 mm. from the limbus and 1.5 to 2 mm. anterior to the insertions of the rectus muscles, to which they bear a definite relationship.

Clinically, these hyaline areas probably escape notice because the patient rarely draws the physician's attention to their presence. I believe that they would be found much more frequently if ophthalmologists paid more attention to such conditions during their routine clinical examinations. It is surprising that so few contributions to the literature on the subject have been made.

REVIEW OF LITERATURE

I have made what I believe to be a complete review of the literature on the subject.

Parsons [1] mentioned hyaline degeneration of the sclera, stating that "hyaline degeneration of the scleral laminae is found under pinguecula, and in other degenerative conditions." He also mentioned fatty degeneration and calcification of the sclera, occurring in advanced age after old inflammatory conditions.

Salzmann [2] mentioned only a fatty degeneration which becomes apparent over the entire tunica fibrosa in old age, manifested on the cornea as arcus senilis, and which causes the sclera to change from a pure white to a yellowish tint. The sclera in old age, he said, becomes generally thickened, more rigid and less elastic.

Rolandi [3] reported 3 cases in 1915, in all of which symmetric changes occurred in the sclera of both eyes. He stated that so far as he knew nothing similar had ever previously been described. In a 76 year old woman, he observed two slate gray spots on each eye, situated between the limbus and the insertions of the external and internal rectus muscles. The spots were semitransparent, nearly glassy, and measured about 5 mm. vertically and 2 mm. horizontally. He could see the fibrous structure of the tendon of the right external rectus muscle through the thin conjunctiva, as well as the insertion of the muscle itself. He attempted to remove a small piece from one of the spots by means of a trephine through an incision in the conjunctiva but was unsuccessful. He said that the trephine met with considerable resistance. Soon after he observed the condition in his first patient, he noted a similar spot on the nasal side of one eye in a 72 year old patient and two large spots on the nasal side of both eyes in a 77 year old patient. Rolandi believed the condition to be a degenerative process. He felt that the slate gray spots were due to mechanical factors, that there followed a transformation of the fibrous tissues of the sclera into cartilage and that the cartilage later degenerated into hyalin, which permitted the pigment of the choroid to shimmer through. He noted the following characteristics with regard to the spots: their elective position, rectangular shape, slate gray color, semitransparency and nearly osseous consistency. Rolandi accompanied his article with photographs which present the same characteristics as those in my cases and in the cases reported in the literature which are cited here.

Krekeler,[4] in describing the structure of the sclera at various periods of life, stated that in advanced age the elastic fibers, especially in the middle layers of the sclera, and the number of

From the Dartmouth Eye Institute, Dartmouth Medical School, Hanover, N. H.

1. Parsons, J. H.: The Pathology of the Eye, London, Hodder & Stoughton, 1904, vol. 1, pt. 1, p. 279.

2. Salzmann, M.: Anatomie und Histologie des menschlichen Augapfels in Normalzustande, seine Entwicklung und sein Altern, Vienna, F. Deuticke, 1912.

3. Rolandi, S.: Sulla presenza di speciali chiazza nella sclera di probabile natura cartilaginea, Ann. di ottal. **44**:843-857, 1915.

4. Krekeler, F.: Die Struktur der Sklera in den verschiedenen Lebensaltern, Arch. f. Augenh. **93**:144-159, 1923.

scleral fibers themselves decrease and that there are deposits of fat globules and lime.

Pillat [5] described, in 1933, a peculiar senile degeneration of the sclera which he had observed in an 88 year old woman. He noted two dark vertical spots in each eye, in front of the insertions of the external and internal rectus muscles. He was also able to see the tendons of the muscles themselves, shining through the thin conjunctiva. Pillat was evidently familiar with the experiments of Fischer,[6] who had observed similar spots in subluxated rabbit eyes, and concluded that the semitransparency and thinness of the sclera in the affected areas were due to loss of the water content of the sclera. Knowing that the entrance of the tendon fibers into the sclera continually expose it to mechanical action, he surmised that the affected area was predisposed to give off more water to offset its mechanical vulnerability. Pillat was familiar with the cases reported by Rolandi but did not believe they were similar to his. He felt that the condition in Rolandi's cases was due to secondary scleral degeneration of some other origin.

Kiss [7] reported a case of senile thinning of the sclera before a session of the Ophthalmological Society in Vienna, on Oct. 16, 1933. In an 86 year old patient he had observed bilateral slate-gray spots in the sclera, situated between the limbus and the insertion of the external rectus muscle. He regarded the condition as an early stage of senile degeneration and inferred that his case was similar to the one described by Pillat. He was sure that the condition was not rare, as he had observed 2 other cases within a few weeks.

Kreibig,[8] in discussing the case reported by Kiss, stated that he had observed a number of patients with similar changes and that only the aged were affected.

Graves has written four times on the condition since 1936. In Berens' textbook, he [9] described a condition which at first glance suggested that a tenotomy of the internal rectus muscle had previously been performed because of a small, sharply defined area where the sclera was thinned.

In 1937, he [10] reported in more detail on cases in which he had observed the condition. At that time he regarded the defects as developmental in character and stated he was not aware that such a condition had previously been reported.

In a later article, Graves [11] said that Ida Mann had questioned whether the defects he had noticed were developmental in character and had suggested that the condition might be due to a hyaline degeneration of the sclera, and not to structural thinning, as he had stated. In this article, Graves said that he had jumped to the conclusion, in the first few cases he had observed, that the condition was a developmental defect but that he was now in agreement with the conclusive and interesting histopathologic evidence described by Culler.[12] Up to this time Graves had observed the condition only on the mesial aspect of the sclera, whereas in the case he was now reporting there were not only mesial plaques but some evidence of lateral plaques.

Graves [13] reported a case in 1941 in which the plaques were seen only on the temporal aspect of each eye. He was now convinced that the condition was not necessarily confined to the mesial aspect of the sclera and agreed that the words "developmental" and "mesial" should be dropped. He felt that the term "scleral plaques" was quite suitable.

Urrets Zavalia and associates [14] described a case of the defect in a man aged 68, which was observed during the course of chronic porphyrinuria. Symmetric bilateral temporal lesions were present. A piece of the degenerative plaque in the left eye was removed for histologic study. The tissue was described as being similar to that of the sclera, but without cellular elements and with hyaline degeneration and calcification. It was felt that the term "scleromalacia" adequately described the condition and that the case was similar to the one described

5. Pillat, A.: Ueber eine eigenartige senile Entartung der Lederhaut an den Ansatzstellen der geraden Augenmuskeln, Ztschr. f. Augenh. **82**:113-123 (Dec.) 1933.

6. Fischer, F. P.: Experimentelle Untersuchungen an der Lederhaut, Arch. f. Augenh. **97**:467-492 (Dec.) 1926.

7. Kiss, J.: Fall von seniler Skleraverdünnung, Klin. Monatsbl. f. Augenh. **92**:121-122 (Jan.) 1934.

8. Kreibig, W., in discussion on Kiss,[7] p. 122.

9. Graves, B., in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 468.

10. Graves, B.: Bilateral Mesial Superficial Deficiency of the Sclera, Brit. J. Ophth. **21**:534-538 (Oct.) 1937.

11. Graves, B.: Bilateral Mesial Superficial "Deficiency" of the Sclera (Scleral Plaques), Brit. J. Ophth. **23**:191-204 (March) 1939.

12. Culler, A. M.: The Pathology of Scleral Plaques: Report of Five Cases of Degenerative Plaques in the Sclera Mesially, One Studied Histologically, Brit. J. Ophth. **23**:44-50 (Jan.) 1939.

13. Graves, B.: Bilateral (Mesial) Deficiency of the Sclera: Scleral Plaques, Brit. J. Ophth. **25**:35-38 (Jan.) 1941.

14. Urrets Zavalia, A.; Maldonado Allende, I., and Obregón Oliva, R.: Scleromalacia Observed During the Course of a Chronic Porphyrinuria, Arch. de oftal. de Buenos Aires **12**:115-137 (March) 1937.

by van der Hoeve,[15] for even though perforation had not occurred, it might logically be expected in the future. The authors wondered whether some relation might exist between the disturbance of the sclera they had observed in their patient and the chronic porphyrinuria, with its peculiar cutaneous lesions. They also wondered whether the ocular condition might have some relation to the curious anomaly of nutrition which was present in their patient. The case described by these authors was undoubtedly one of senile hyaline scleral plaques, and the accompanying chronic porphyrinuria and nutritional disturbances were purely coincidental.

Gasteiger,[16] in 1937, reported a case of senile degeneration of the sclera at the insertions of the rectus muscles in a 78 year old woman. The bandlike changes were noted both temporally and nasally in each eye. A narrow, horizontal band was seen in front of the insertion of the inferior rectus muscle in the right eye only. Gasteiger expressed agreement with Pillat that the condition was the result of dehydration and that the process first becomes conspicuous in the areas of the insertions of the muscles, as they are especially vulnerable to damage of this sort. By treating pigs' eyes with glycerin and alcohol, he produced dehydration, through fluid drainage, within a few minutes, but the condition of the eyes was normal after they emerged from water or a saline solution. He produced similar changes in the eyes of living rabbits. Under the influence of the normal tissue fluids, however, the sclera quickly reverted to its natural condition. Since Gasteiger was unable to make the bands in his patient's eyes disappear by touching them with salt or by irrigation, he concluded that, in addition to dehydration, there must be permanent changes in the tissues, which might be designated as a form of senile atrophy.

Rones,[17] in a comprehensive survey of senile changes in and degenerations of the eye, included the clinical entity described by van der Hoeve[15] of "degenerative hole-formation in the sclera without evidence of inflammation" but did not mention senile hyaline scleral plaques. He stated that senile changes of the sclera are characterized by fatty infiltration, frequent deposits of fine granules of calcium salts in the posterior segment and widening of the connective tissue bundles with diminution in the number of nuclei. He stated, further, that degeneration of elastic tissue occurs, that the vessels become surrounded by fat droplets and that the arteries particularly show evidence of sclerosis. Rones stated, further, that the sclera is not frequently involved in degenerative changes of old age and that occasionally calcification, rarely bone formation, associated with inflammatory symptoms, is seen.

Culler[12] reported 5 cases of scleral plaques in 1939. All of the lesions in his cases were located mesially. He was able to obtain a specimen at autopsy in 1 of his cases and described the histologic appearance as clearly a degenerative change, involving large masses of hyalin. He permitted me to study six excellent Kodachrome slides illustrating his cases. The clinical appearance of the plaques in his cases was identical with that of the plaques in my cases.

Kyrieleis,[18] in 1939, reported certain changes of the sclera in 3 patients of advanced age. A 77 year old man, who had had one eye enucleated, showed a thinning of the sclera of the other eye both nasally and temporally, not far from the limbus, through which the uvea was discernible. A second patient, aged 70, presented the same type of scleral thinning in both eyes, but only on the nasal side. In the third patient, a 60 year old man, the lesions were bilateral and were seen nasally as well as temporally. Kyrieleis had the opportunity to study the enucleated eye of his first patient. The eye had been removed because of absolute glaucoma due to a melanosarcoma of the choroid. Scleral lesions were present in this eye and appeared much like those in the remaining eye. Histologic examination showed degeneration of the superficial fibers of the sclera and pronounced calcification. Kyrieleis acknowledged that the histologic picture was complicated by the fact that the specimen came from a glaucomatous eye; at the same time, he pointed out that in the case described by Urrets Zavalia and associates[14] some calcification, in addition to hyaline degeneration, was present. While Kyrieleis regarded the lesion he had observed as circumscribed degeneration of the sclera (scleromalacia), he expressed doubt that it was basically different from the lesion known as scleromalacia perforans. He stated the opinion that the calcium plaques within the scleral tissue were virtually sequestrated and would eventually perforate, thus reaching the stage of the condition described by

15. van der Hoeve, J.: Scleromalacia Perforans, Arch. Ophth. **11**:111-118 (Jan.) 1934.

16. Gasteiger, H.: Ueber senile Entartung der Lederhaut an den Ansatzstellen der Augenmuskeln, Klin. Monatsbl. f. Augenh. **98**:767-772 (June) 1937.

17. Rones, B.: Senile Changes and Degenerations of the Human Eye, Am. J. Ophth. **21**:239-255 (March) 1938.

18. Kyrieleis, W.: Ueber umschriebenen Lederhautschwund (Skleromalazie) in höherem Lebensalter, Klin. Monatsbl. f. Augenh. **103**:441-452 (Oct.-Nov.) 1939.

van der Hoeve.[15] He also called attention to the fact that in his cases and in the case described by Urrets Zavalia and associates the spots were regularly located, while in other cases of scleromalacia reported the areas were scattered indiscriminately.

Summarizing the studies made on his own patients, Kyrieleis stated that clinically the lesions appeared as degenerative changes in the sclera. They were located between the insertions of the horizontal rectus muscles and the limbus. He was unable to ascertain in any of his cases when the areas first appeared. In none of his cases had the lesion reached the stage of perforation. Two of his patients had an associated arthritic condition, and he inferred that rheumatic disease favored the development of the scleral defects. He also stated the belief that senile changes of metabolism and of the blood vessels were significant.

Duke-Elder[19] made no mention of senile hyaline scleral plaques in his textbook, although he specifically mentioned the condition recently described by van der Hoeve[15] as "scleromalacia perforans." Duke-Elder stated that degenerative changes are not uncommon in the sclera and that fatty degeneration is usual in old age, the process giving the sclera a yellowish color. He stated that calcification occurs not uncommonly both as a senile degeneration and as a terminal event in postinflammatory fibrosis. As for hyaline degeneration, he merely stated that it is almost universally found in old age, in the tissues under a pinguecula, as a sequel to inflammation and by extension from the subconjunctival tissues. Senile hyaline scleral plaques can hardly be included under such a description.

Von Bürki,[20] in his review of diseases of the sclera, mentioned the cases of senile degeneration of the sclera described by Gasteiger,[16] Culler[12] and Graves.[21]

Boshoff,[22] in 1942, described observations in 11 cases of scleral plaques and discussed the literature. He gave credit to Pillat[5] for having given the first full description of the condition and to Culler[12] for the first histologic description of scleral plaques.

I have already brought out in this review that Rolandi,[3] eighteen years before Pillat, described degenerative changes in the sclera of the eyes of 3 patients, both eyes being affected in each case. He accompanied his description with photographs clearly showing that the condition was identical with that in the cases described here. Pillat himself was familiar with the cases reported by Rolandi, but, as I have said before, he did not believe that the defects were of the same nature as the lesions in his cases. I have also shown that Urrets Zavalia and associates,[14] two years before Culler,[12] reported histologic studies in a case of scleral plaques.

Although only 4 of his 11 patients had arthritis, Boshoff[22] stated that there seemed to be a definite connection between polyarthritic disease and scleral hyaline plaques. He was unable to discover any other constitutional dyscrasia which might have had an influence on the formation of the plaques. Boshoff supported Culler and Graves in their choice of the name "scleral plaques," which he said described the essential nature of the condition. He[22] mentioned in a footnote that Ascher described the condition at an ophthalmologic congress in Vienna at about the same time as Pillat. Ascher[23] does not recall the exact date of his discussion mentioned by Boshoff and makes no claim to priority in describing the condition. He became interested in these scleral spots after learning of the experiments of Kahn and Fischer.[6] He expressed the belief that there might be some connection between these experiments on dehydration and the scleral spots of hyalinization. Ascher states that he rarely saw these scleral spots in Prague but that he has observed them more frequently in this country. He observed 4 patients in the nutrition clinic at Birmingham, Ala., all of whom had nicotinic acid deficiency, which in 1 patient was combined with a deficiency in riboflavin and thiamine. Ascher raises the question whether such deficiencies or the hot climate or both may have some influence on the development of the scleral hyaline spots. He has never observed such a spot in relation to the superior rectus muscle and offers the explanation that this part of the sclera is constantly covered by the upper lid and protected against noxious factors, one of them possibly being exsiccation. Ascher also points out that the fibers of the tendons of both the external and the internal rectus muscle are visible with oblique illumination but are still clearer with the corneal microscope.

ANATOMIC AND PHYSIOLOGIC CHARACTERISTICS OF THE SCLERA

An understanding of the structure and physiologic activity of the sclera enables one to appreciate how ideally it is protected against the

19. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1940, vol. 2, p. 2065.

20. von Bürki, E.: Cornea, Episklera, Sklera, Ophthalmologica **103**:405-420, 1942.

21. Graves (footnotes 9, 10, 11 and 13).

22. Boshoff, P. H.: Hyaline Scleral Plaques, Arch. Ophth. **28**:503-506 (Sept.) 1942.

23. Ascher, K. W.: Personal communication to the author.

invasion of micro-organisms and yet how peculiarly defenseless it is to repel infection or to combat influences making for degeneration.

The sclera consists of fibrils of connective tissue united into bundles, resembling the lamellas of the cornea but less regular and more firmly packed. The bundles run chiefly in two directions, meridionally and equatorially. There are also many oblique bundles. The entire arrangement is interrupted by the entrance of the tendons of the extraocular muscles. A large number of extremely fine elastic fibers are scattered throughout. They are fewer in youth. The outer layers of the sclera contain more elastic fibers than the inner, and the fibers are more numerous posteriorly than anteriorly. Between the bundles of fibers are lymph spaces, containing fixed connective tissue cells, though these are far less numerous than in the cornea. The inner surface of the sclera is normally pigmented and forms the lamina fusca. It is united with the choroid by fibrils of pigmented connective tissue and is lined with endothelial cells, thus forming the external wall of the suprachoroidal lymph space. The outer surface of the sclera is also covered with a single layer of endothelial cells.

The sclera, being composed almost entirely of fibrous and elastic tissue and with merely a protective function to perform, carries on a very low grade of metabolic activity and is therefore poorly vascularized. The blood vessels and nerves of the sclera are few. Arteries are given off from the short posterior and anterior ciliary vessels. The posterior ciliary veins—smaller than the corresponding arteries—are derived entirely from the sclera, the anterior ciliary veins only in part. The blood vessels form a network almost entirely confined to the episcleral tissues. This network is extremely attenuated except in the anterior region near the corneal margin. The close capillary system here may be seen clinically in inflammatory conditions as it enters into the picture of a ciliary injection.

The nerve supply of the sclera is scant and is derived from the posterior ciliary nerves as they run between the sclera and the choroid.

The scarcity of blood supply in the sclera accounts in part for the slow healing of scleral wounds. The extraordinarily low regenerative ability of scleral tissue, which is partly due to the scarcity of blood in the sclera, has great practical significance in ocular surgical procedures involving the sclera. The results of trephining operations on the sclera for the purpose of lowering the intraocular tension in cases of glaucoma are determined by this factor. When filtration ceases, it is not the result of scar formation but is due to filling in of the opening by a substitute tissue, which is more compact than normal scleral tissue and which arises from the episclera and uvea—the sclera playing little part in its formation. The resulting scar has transparency but is not permeable to fluids.

The opaque white appearance of the sclera depends on its water content. Studies of the chemical composition of the sclera have shown that it contains a large percentage of water—over 65 per cent—while proteins make up the bulk of the solid matter.

Fischer [6] has shown that the fibers constitute a gel which combines reversibly with water, and that when the water content is at its normal level, it reflects the light diffusely so that it appears opaque. Fischer demonstrated that with a fluid content of 40 to 80 per cent the sclera of rabbits remained white, while with an increase or decrease of fluid content it became semitransparent.

Fischer credited Kahn with first observing the appearance of rectangular spots in the region of the insertion of the rectus muscles in subluxated rabbit eyes. Fischer proved that the spots thus produced are the result of local dehydration, and are in no way produced by any changes in circulation, intraocular tension, undue stretching of ocular muscles or nervous factors, and that the phenomenon is reversible. He discovered that the phenomenon is not limited to such specific locations but can be produced over the entire sclera by the use of strong hygroscopic materials, and that it is also reversible for the entire sclera. He further proved that the sclera becomes transparent not only when it is dehydrated but when its fluid content is above normal. In histologic studies, Fischer observed only marked thinning of the sclera that had been dehydrated and so could give no explanation as to why transparency exists in the spots produced. The question is much the same as that which has so long fascinated histologists with reference to the cornea and its transparency. Although the experimental spots induced by Fischer disappeared on replacement of the globe, no local measure has been known to change in any manner the appearance of the spots observed in patients of advanced years.

ETIOLOGIC FACTORS

Advanced age is largely responsible for predisposition to the disease, since in the aged dehydration and progressive sclerosis of the connective tissue of the entire body, as well as of the external tunic of the eye, occur. It is probably the result of a local nutritional disturbance, which, in turn, is due to arteriosclerotic changes. Certainly, vascular changes must be of funda-

mental importance, since degeneration taken as a whole is the result of faulty circulation.

The fact that this unusual degenerative condition of the sclera has a predilection for the area lying in front of the insertions of the rectus muscles leads one to the additional assumption that the changes occur not only because of local circulatory disturbances but because of the constant stress and strain on the scleral fibers which are in such close proximity to the insertions of the rectus muscles.

Susceptible as the elderly are to some form of arthritis in peripheral joints, the spinal column or both, the presence of aggravated or pronounced arthritis in a patient with senile hyaline scleral plaques should not lead one to assume that the ocular lesion is essentially a part of the disease. I am not convinced of any definite connection between polyarthritic disease and senile hyaline scleral plaques. A rheumatic disorder might well aggravate an existing vasosclerotic condition and so be an indirect contributory factor. In reviewing nearly 50 cases reported in the literature, together with my own 5 cases, I have been unable to determine any single constitutional disease which, either directly or indirectly, influences the formation of the plaques.

CLINICAL DESCRIPTION

Senile hyaline scleral plaques must be regarded as a clinical entity, although the condition is characterized by absence of clinical symptoms.

The plaques are situated about 3 mm. from the limbus and 1.5 mm. to 2 mm. anterior to the insertions of the rectus muscles. They are generally rectangular, but occasionally only a rounded or oval patch, situated opposite either end of the insertion of a rectus muscle, is observed. They are seen most commonly opposite the insertion of the internal rectus muscle but occur next in frequency opposite that of the external rectus muscle. Rarely have they been observed opposite the insertion of the inferior rectus muscle. In none of my cases, nor in any of the cases described in the literature, were plaques observed opposite the insertion of the superior rectus muscle. Their average width is 1.5 mm. to 2 mm. and their average length 5 mm. to 6 mm. The patches are not elevated but appear somewhat depressed. The limbal margins in general are more sharply defined. The irregularity of the tendinous margins is probably due to the presence of the interwoven tendon fibers. The sclera does not appear to have lost any of its firmness; in fact, it seems to be even more rigid and resistant. On transillumination of the globe, the areas show as relatively clear and translucent windows.

The spots do not correspond to the muscle insertion themselves but lie just ahead of them, favoring the insertions a little more than the limbal margins. In many instances, one is able to observe through the thin conjunctiva an individual muscle tendon and its insertion as well, the tendon ending in a perpendicular line close to the spot. The slight obliquity of the dark bands also conforms to the slightly oblique insertions of the horizontal rectus muscles. The length of the dark bands also corresponds fairly well to the width of the tendon insertions.

There is no disturbance of the overlying conjunctiva. Episcleral and conjunctival blood vessels course normally over the patches except for occasional tortuosity in keeping with the vasosclerotic state of the patient.

The slate gray discoloration has nothing to do with pigmentation of the sclera, the episclera or the bulbar conjunctiva. When the lesions are studied carefully with oblique illumination and a loupe or a slit lamp and corneal microscope, the dark discoloration is seen as a shining of the uveal pigment of the ciliary body through the semitransparent sclera in that area. Even with the naked eye one can see that the peculiar slate gray color bears no resemblance to the brownish yellow tint of a pigmentation.

As far as I have been able to determine, the condition is not a hazard to the eye. When present, lowered visual acuity was due to changes either in the lens or in the fundus or to chronic glaucoma, not to the plaques.

HISTOLOGIC DESCRIPTION

Histologic studies have shown that the condition is primarily degenerative. In place of normal sclera, there remain some cellular and nuclear fragments and large masses of hyalin. There is general atrophy of the overlying conjunctiva and episclera.

Marked calcification was reported in 2 cases. In 1 instance (Kyrieleis [18]) the picture was complicated by the fact that the eye was glaucomatous. In the other, the patient was suffering with a curious anomaly of nutrition (Urrets Zavalia and associates [14]).

DIFFERENTIAL DIAGNOSIS

Scleromalacia perforans.—A condition which bears no real resemblance, but which has been confused with it, is scleromalacia perforans. The two conditions are alike only in their chronicity and in the fact that they affect the sclera predominantly. Scleromalacia perforans is primarily necrotic in origin, whereas senile hyaline scleral plaques are primarily degenerative in character.

Culler [12] questioned whether these scleral spots were not the early stage of scleromalacia perforans. Kyrieleis [18] expressed doubt that the condition in his 3 cases was basically different from scleromalacia perforans and stated the belief that the plaques would eventually perforate, thus reaching the stage of the condition described by van der Hoeve.[15] Urrets Zavalia and associates [14] applied only the term scleromalacia, predicting that in their case the patches would perforate later.

Verhoeff and King [24] evolved a description of the clinical course of scleromalacia perforans which distinguishes this condition from that of senile hyaline scleral plaques. Scleromalacia perforans starts with slightly elevated nodules, involving the sclera and the overlying tissue. These nodules may be situated anywhere between the corneal limbus and the equator. Definite evidence of rheumatoid arthritis exists at the onset of the scleritis. Necrotic processes follow, resulting in formation of a sequestrum, which becomes completely disintegrated and densely infiltrated with necrotic pus cells. A cavity results, due to perforation and discharge of the sequestrum.

In cases of senile hyaline scleral plaques, a history of any serious arthritis is lacking, nor is there a history of nodules on the sclera. The lesions have not been known to occur anywhere except between the insertions of the internal or the external rectus muscle and the inferior rectus muscle and the limbus, whereas scleromalacia perforans may occur anywhere in the sclera which is covered by conjunctiva.

Brawny Scleritis.—While brawny scleritis affects the sclera predominantly and occurs in the advanced years of life, it differs greatly histologically from senile hyaline scleral plaques. In brawny scleritis there are an invasion of the anterior portion of the sclera and the tendons of the rectus muscles with granulation tissue richly infiltrated with plasma cells and a diffuse plasma cell infiltration of the ciliary body and the anterior part of the choroid (Verhoeff [25]).

Blue Sclera.—Needless to say, blue sclera does not have to be differentiated from senile hyaline scleral plaques, which is a degenerative change, and not a developmental one.

Sarcoma.—A senile hyaline scleral plaque must not be mistaken for a sarcoma, and enucleation should never be considered. Graves [10] mentioned that in 1 of his cases the patient had consulted him because she had been told that her eye was sarcomatous. On careful inspection of the defect, the clearcut appearance of senile hyaline scleral plaques does not suggest a disintegrating eruption from beneath. The slate gray color of the plaque, moreover, has nothing whatever to do with pigmentation.

24. Verhoeff, F. H., and King, M. J.: Scleromalacia Perforans: Report of a Case in Which the Eye Was Examined Microscopically, Arch. Ophth. **20**:1013-1035 (Dec.) 1938.

25. Verhoeff, F. H.: Brawny Scleritis, Ophthalmoscope **11**:2-10, 1913.

REPORT OF CASES

CASE 1.—Mrs. W. C., aged 81, who was first seen on July 22, 1944, complained of blurred vision. Vision in the right eye had been poor since an attack of iritis and ulcers, fifteen years before. Vision in the left eye was satisfactory. The patient was able to read newsprint without a magnifying glass. There were no known chronic ailments. Corrected vision was 20/70—2 in the right eye and 20/40+2 in the left eye. An immature senile cortical cataract was present in the right eye and another, less advanced, in the left eye. The right fundus was blurred; arteriosclerotic changes with accompanying retinopathy were noted in the left eye. Examination with the slit lamp revealed evidence of old anterior uveitis in the right eye. Intraocular tension was 22 mm. in the right eye and 25 mm. in the left eye (Schiøtz).

On the mesial aspect of each eye, slate gray plaques were noted. They were situated about 3 mm. from the limbus and about 1.5 to 2 mm. anterior to the insertion of the internal rectus muscle. Their average width was 2 mm. and their length 5 to 6 mm. (fig. 1 *A* and *B*).

On the temporal aspect of each eye was seen a somewhat rounded, slate gray patch, situated between the insertion of the external rectus muscle and the limbus (fig. 1 *C* and *D*).

While the patient and her daughter were both aware of these spots, they were unable to state when they had first noticed them. The oculist who had attended the patient for many years had observed the dark areas on the sclera but was also unable to state when he had first noticed them.

There was no disturbance of the overlying conjunctiva. On transillumination of the globe, the areas appeared as relatively clear, translucent windows. The several tortuous conjunctival vessels seen in each eye were in keeping with the age and vasosclerotic state of the patient.

The patches were not elevated but were somewhat depressed. The edges were more sharply defined along the corneal border. When they were viewed with oblique illumination and a loupe or a slit lamp and corneal microscope, it was clearly evident that the spots were not pigmented but were the result of thinning of the sclera itself. Beneath the glassy patches the uveal pigment of the ciliary body was visible.

CASE 2.—Mr. W. G., aged 78, was first seen on Oct. 10, 1944. His poor vision in both eyes was due to chronic glaucoma of long standing. Visual acuity was 20/80 in the right eye and 20/200 in the left eye. The visual fields of both eyes were constricted to within 10 degrees of fixation except for a narrow projection below to the 25 degree isopter. The tension in each eye averaged 19 to 25 mm. (Schiøtz). The patient was using 1 per cent pilocarpine nitrate three times a day. He had no chronic ailment and had always been exceptionally well.

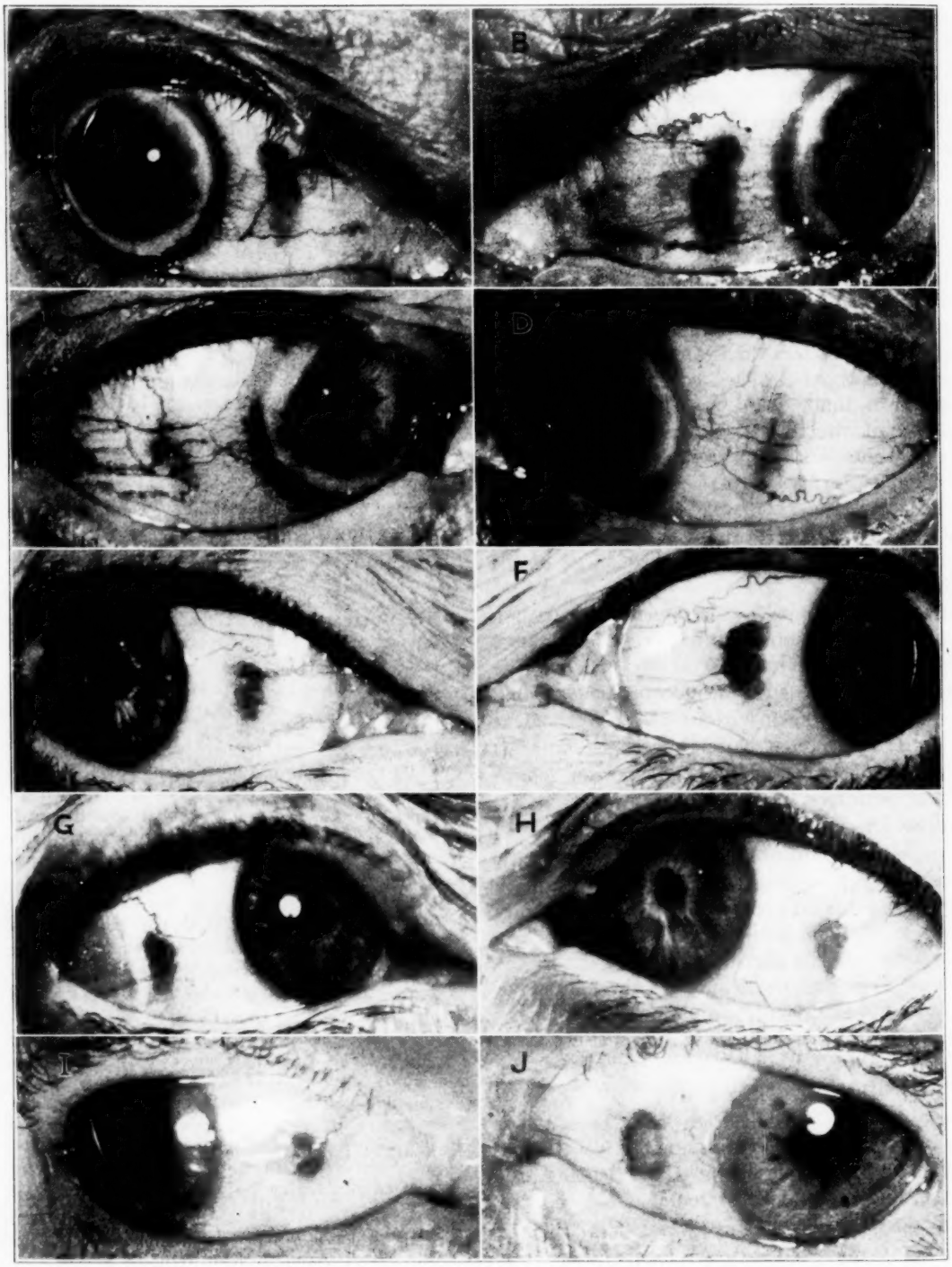


Fig. 1.—Case 1: *A*, mesial aspect of right eye; *B*, mesial aspect of left eye; *C*, temporal aspect of right eye; *D*, temporal aspect of left eye. Case 3: *E*, mesial aspect of right eye; *F*, mesial aspect of left eye; *G*, temporal aspect of right eye; *H*, temporal aspect of left eye. Case 4: *I*, mesial aspect of right eye; *J*, mesial aspect of left eye.

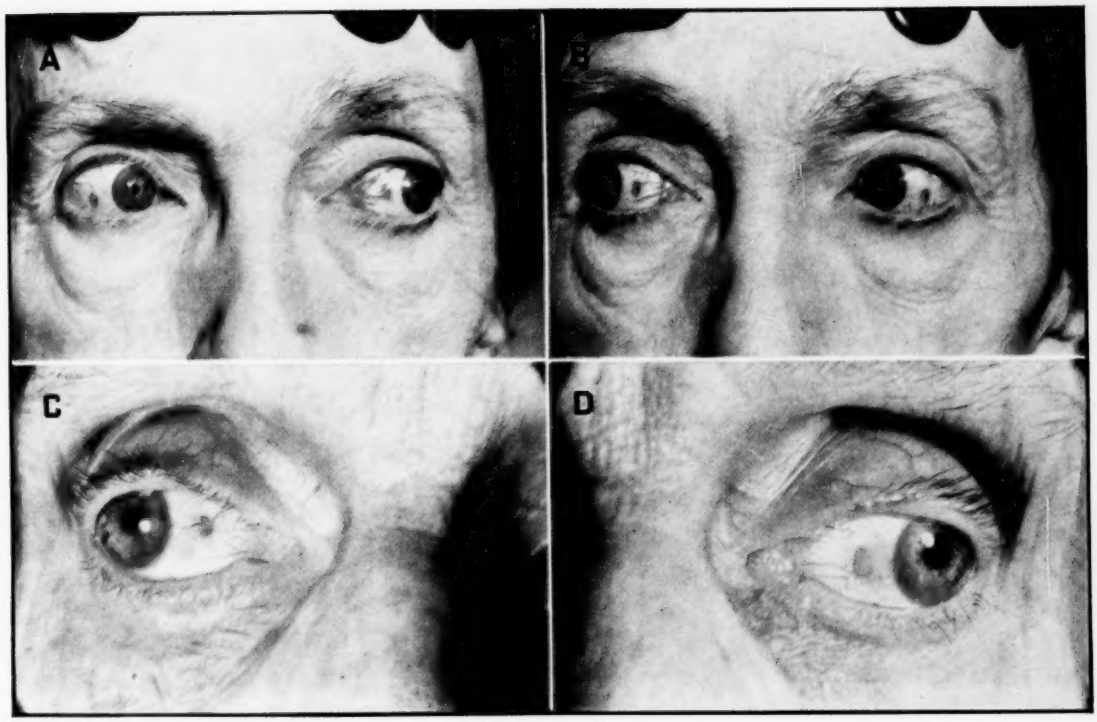


Fig. 2.—Case 3: *A*, patient looking to left; *B*, patient looking to right. Case 4: *C*, mesial aspect of right eye; *D*, mesial aspect of left eye.

Slate gray plaques were noted on both the mesial and the temporal aspect of each eye; they were most marked on the temporal aspect of the left eye and least marked on the nasal side of the right eye.

CASE 3.—Mrs. F. M., aged 72, was first seen on Nov. 6, 1944. She complained of dryness and sticking of the lids, especially on retiring and on awakening in the morning. She had had an attack of arthritis in her left shoulder thirty years previously and a nasal operation and cholecystectomy in recent years. The refractive error was hyperopic astigmatism and presbyopia. Corrected vision was 20/20 in each eye.

When the patient's attention was called to the presence of the slate gray patches on both her eyes, she said that she had been aware of them for many years. The plaques were rectangular and were situated anterior to the insertions of the internal and external rectus muscles of each eye. The plaque on the lateral aspect of the right eye was 1.75 mm. wide and 4 mm. long, and the plaque on the mesial aspect measured approximately 2 by 3.5 mm. The plaque on the mesial aspect of the left eye measured 2.5 by 5 mm., and that on the lateral aspect, 1 by 3 mm. (figs. 1 *E*, *F*, *G* and *H* and 2 *A* and *B*).

The tendons of both external rectus muscles, and their insertions as well, were clearly visible through the conjunctiva. The insertions ended in perpendicular lines close to the plaques. The tendons and insertions of the internal rectus muscles could also be seen but were somewhat obscured by overlying conjunctival vessels.

CASE 4.—Mrs. M. A., aged 88, was first seen on Nov. 22, 1944. She complained of failing vision in both eyes for thirty-five years. For several years she had been using a magnifying glass for reading and had been seeing halos around lights. Her vision had seemed worse and the halos more pronounced during the past week. She was found to have moderate hypertension and auricular fibrillation. She had been troubled with arthritis of the left knee for the past ten years.

Visual acuity was 20/400 in each eye and was not improved with glasses. The patient had an immature senile nuclear cataract and senile macular degeneration in each eye. The intraocular tension was 21 mm. in the right eye and 19 mm. in the left eye (Schiøtz).

A slate gray plaque was noted on the mesial aspect of each eye (figs. 1 *I* and *J* and 2 *C* and *D*). Each plaque was situated about 3 mm. from the limbus and anterior to the insertion of the internal rectus muscle. The patient was not aware of their presence.

CASE 5.—Mrs. E. F., aged 72, was first seen on Dec. 6, 1944. She complained of failing vision in both eyes. She had a high degree of myopia and had worn glasses since 11 years of age. She had been aware of spots on the inner side of each eye for several years. Her general health was good.

Corrected vision was 20/25—2 in the right eye and 20/200 in the left eye. Myopic choroiditis was present in both eyes.

Slate gray plaques were noted on both the mesial and the temporal aspect of each eye. The plaque on the mesial aspect of the right eye measured approximately 1.5 by 4 mm. Only a rounded patch was noted on the mesial aspect of the left eye. It was situated opposite the lower end of the insertion of the left internal rectus muscle.

Rounded patches were faintly visible on the temporal aspect of each eye, opposite the insertion of the external rectus muscle. The fibers of the external rectus muscles could be faintly seen through the conjunctiva, as could their insertions, which ended in a perpendicular line close to the spots.

SUMMARY AND CONCLUSIONS

A review of the literature concerning the clinical entity which I term "senile hyaline scleral plaques" is presented. In addition to the cases reviewed from the literature, I report 5 cases of my own.

It is my belief that this interesting condition of the sclera would be found more frequently if ophthalmologists were on the alert for such a defect during their routine clinical examinations. Patients are frequently not aware of the presence of the plaques.

The terms "bilateral mesial superficial 'deficiency' of the sclera (scleral plaques)" (Graves [11]), "scleral plaques" (Culler [12]) and "hyaline scleral plaques" (Boshoff [22]) all fail to indicate one important feature of the disease. Since advanced age is probably the most important predisposing factor in the disease, it is my opinion that the term "senile hyaline scleral plaques" describes the disease more completely and accurately.

58 East Washington Street.

TRAUMATIC IRIDODIALYSIS: ITS SURGICAL CORRECTION

REPORT OF A CASE

AARON BARLOW, M.D., AND HERMAN L. WEINER, M.D.
PHILADELPHIA

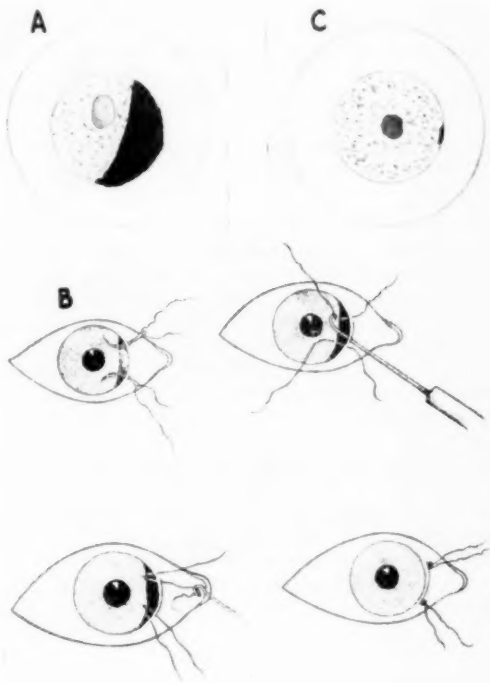
A direct blow on the eye will sometimes cause a detachment of the root of the iris from its ciliary insertion—traumatic iridodialysis. When the separation is small, it is of no concern either from a visual or a cosmetic viewpoint. Frequent instillations of atropine and protection from light by dark glasses or a bandage may result in a reattachment, according to some authors. However, when the separation is large, it may cause monocular diplopia, owing to a double pupil. But of even graver concern in some cases is the unsightly cosmetic effect produced by the distortion of the normal contour of the iris and pupil. When the victim of the accident is a little girl, as in the following case, one has a strong desire to do something to correct the deformity.

REPORT OF CASE

A girl aged 6 was struck in the left eye with a BB shot while playing in the street. She was admitted to the Mount Sinai Hospital on April 28, 1943, soon after the accident. The eye was intensely injected, and the anterior chamber was filled with blood. The temporal base of the iris, from 1 to 6 o'clock, was torn away from its ciliary attachment, forming a large separation, about 5 mm. wide in the horizontal meridian. The pupil was distorted and drawn nasally and upward. The fundus could not be seen because of the hemorrhage in the anterior chamber. In treatment atropine, an antiseptic collyrium and sterile dressings were used. As the hyphemia gradually cleared, a fairly good view of the fundus could be obtained through the pupil, but not through the dialysis. Apparently, the rupture caused a separation of the stroma of the iris from its underlying pigment layer. The pigment layer thus prevented a view of the fundus through the dialysis. Later the lens also became opaque, forming a traumatic cataract (fig. *A*).

The following ingenious operation, with its gratifying result, was devised by Jameson and has been described by Wiener and Alvis.[1] Jameson's original article, with a report of 4 cases, was published in 1909 in the ARCHIVES OF OPHTHALMOLOGY.[2] The technic, which we closely adhered to, is as follows:

A small, curved eye needle, threaded with fine silk (we used 000000), is introduced in the sclera 2 or 3 mm. behind the limbus above the horizontal line, passed into the anterior chamber, transfixing the base of the torn iris, and passed out through the cornea. The needle is then removed. A similar suture is then passed below the horizontal line. An incision is then made in the limbus with a narrow keratome between the points of entrance of the two needles in the sclera. Through this opening a small, blunt iris hook is introduced; the hook engages that part of the thread between the iris and the cornea, pulling the end of the thread out of the cornea, back into the anterior chamber and through the wound out of the eye. The threads are then tied, gently bringing the torn base of the iris back to the sclerocorneal junction (fig. *B*).



A, diagram showing appearance of the eye before operation; *B*, drawing from Jameson,[2] illustrating technic of operation, and *C*, diagram showing appearance after operation.

This operation was performed on Oct. 10, 1943, about six months after the injury. Atropine was instilled and the eye bandaged. The postoperative course was uneventful. The eye was redressed on the second day and atropine instilled daily thereafter. The sutures were removed on the eighth day, and the child was discharged two days later. Subsequently (about three

Read before the Section on Ophthalmology of the College of Physicians, Philadelphia, March 15, 1945.

1. Wiener, M., and Alvis, B. Y.: Surgery of the Eye, Philadelphia, W. B. Saunders Company, 1939.

2. Jameson, P. C.: Reattachment in Iridodialysis: A Method Which Does Not Incarcerate the Iris, Arch. Ophth. **38**:391, 1909.

months later), on Jan. 13, 1944, the traumatic cataract was removed by a discission and, again, on June 29, by a capsulotomy. The result was a clear, fairly round pupil (fig. *C*). A small, very fine chink between the points of entrance of the two needles is visible on close inspection. The corneal needle punctures were invisible after several months but are distinguishable with the slit lamp. An approximate correction with a 12 D. lens gave 20/70 vision. When the child is older and more cooperative, a careful refraction will probably give better results, since the fundus appears to have escaped serious damage. Vision in the right eye was 20/20.

COMMENT

Reports in the literature on the surgical treatment of traumatic iridodialysis are not numerous. Most of the operators have used the method of incarceration of the iris, either by pulling the base of the iris into the limbal incision or by suturing with one or more sutures a few of the iris fibers to the scleral junction immediately behind the cornea. Bulson [3] reported 3 cases in which the method of incarceration of the iris was used, and Goldfeder [4] reported 2 cases in which the same method was employed, with successful results. Key [5] reported 4 cases in which operation was successfully performed by the suture method. Spaeth [6] describes a similar operation, also based on the principle of incarceration of the iris, which he prefers to do under a conjunctival flap in order to prevent infection from the exposed sutures. However, if the operation is done under aseptic conditions and with a sterile culture, the danger of infection should be no greater than in any other intraocular operation in which sutures are used—cataract extraction, for example. Wheeler [7] looked with disfavor on the method of incarceration of the iris, which, he said, "drags the iris into the limbus wound in such a way as to form an iris prolapse." Neither did he approve of sutures, on the general ground that "sutures should not be passed through eye structures unnecessarily." He maintained that "an attachment can be easily and permanently secured by simply carrying a tiny shred of tissue from the torn edge of the iris very slightly into the limbus wound."

3. Bulson, A. E., Jr.: Correction of Iridodialysis by Operation, Am. J. Ophth. **3**:357, 1920.

4. Goldfeder, A. E.: Ueber die operative Behandlung der Iridodialyse, Klin. Monatsbl. f. Augenh. **89**: 229, 1932.

5. Key, B. W.: Iridodialysis as a Clinical Entity, Am. J. Ophth. **17**:301, 1934.

6. Spaeth, E. B.: Principles and Practice of Ophthalmic Surgery, ed. 3, Philadelphia, Lea & Febiger, 1944.

7. Wheeler, J. M.: Principles of Modern Surgery in Ophthalmology, Am. J. Ophth. **17**: 683, 1934.

The Jameson operation does not incarcerate the iris between the lips of the wound but brings the torn surface in contact with a freshened surface, within the anterior chamber. Reattachment takes place by agglutination through a plastic exudate thrown out from the iris at the point of contact.

We have had no previous experience with the surgical treatment of traumatic iridodialysis, and we do not presume to judge the merits of this or of other methods. In this particular case we were fortunate to obtain the desired result with the method described, and of course we are pleased. At the time of writing, about a year and a half after the operation, there are no signs or symptoms of any late complications, and the iris is still intact. The right eye has remained normal throughout.

In conclusion, we wish to state that we are keenly conscious of the fact that this operation, like any other intraocular procedure, is not without its potential risks and should not be undertaken too lightly. One of the possible danagers is injury of the lens (when the lens is clear) during the operation, thus producing a traumatic cataract. Secondary glaucoma as a possible late complication is mentioned by some authors. However, as far as we know, no case of secondary glaucoma following the surgical correction of traumatic iridodialysis has ever been reported. The operation should not be performed when the dialysis is small or when the defect is concealed by the lids. However, when the defect is large and the cosmetic effect is such that it can be a source of real embarrassment, the operation, we believe, is justified.

SUMMARY

A case of extensive traumatic iridodialysis in a 6 year old girl, successfully corrected by the Jameson operation, is reported.

The technic of the operation, with illustrative drawings, is described in detail.

The child has been under observation for about a year and a half since the operation, and during all that time there have been no signs or symptoms of any late complications.

DISCUSSION

DR. WARREN S. REESE, Philadelphia: It might be possible to avoid a keratome incision by using a self-threading needle and two sutures, one of which is passed between the two strands of the first suture just beyond its exit from the iris. The needles could be removed after perforation of the cornea. The suture could

then be pulled back into the anterior chamber, one acting as a plug and preventing the other from slipping back through the iris.

DR. JAMES S. SHIPMAN, Philadelphia: In reading the technic of this operation, I could not understand why the cornea would not be badly scarred in cases of iridodialysis sufficiently large to justify this operation.

Certainly infection is also a danger, since the needles and sutures pass out through the cornea, and the free suture has again to be pulled back into the anterior chamber. My experience with suturing the cornea has been that one is more likely to get sloughing, with infection about the sutures, than when suturing the sclera.

I do not feel that the conjunctival flap is necessary. On the other hand, one is probably better off without a conjunctival flap, which is likely to dam up the secretions and give rise to more slough about the sutures.

TESTS FOR DETECTION AND ANALYSIS OF COLOR BLINDNESS

I. AN EVALUATION OF THE ISHIHARA TEST

LE GRAND H. HARDY, M.D.; GERTRUDE RAND PH.D.,
AND
M. CATHERINE RITTLER, B.A.
NEW YORK

Because of their great convenience and apparent simplicity of administration, polychromatic charts for the detection of defective color vision have been widely used in military and industrial fields for many years. Much dissatisfaction has been reported, and both the statistics of the Civil Aeronautics Administration and the results obtained by the Color Blindness Committee (Army Air Forces, United States Navy, Civil Aeronautics Administration and Inter-Society Color Council) indicate that, as they are used at present, polychromatic test plates are only about 50 per cent effective in screening out persons with defective color vision by the criterion of the test itself.

In an endeavor to discover the reason for this failure, extensive investigations have been undertaken at the Knapp Memorial Laboratories utilizing a large battery of tests on all types of subjects, ranging from persons with normal to those with extremely defective color vision. Our results indicate that while defects in the tests themselves account for part of the discrepancies, other important sources of error have arisen, namely: (1) disregard of the critical importance of illumination; (2) careless and inefficient administration; (3) incompetent and invalid interpretation of results; (4) misapplication of the test to fields for which they were not intended, or the expectation of more from the tests than they were capable of, or designed for, yielding.

One of us (L. H. H.) has for years insisted on the critical importance of the illumination used for viewing materials seen by reflected light, claiming that as they are at present administered polychromatic tests have become as much tests of the lighting system used as they are tests of the subject's color discriminatory ability.

Likewise, he has asserted that results obtained from polychromatic test plates, with their low values and chromas and large patterns yielding dim and extensive retinal images, must with great caution be applied to situations requiring the recognition of luminous objects which yield small but bright retinal images. In the absence of time and facilities for carrying out complete colorimetric investigations, at least two types of tests should be given a subject before a complete diagnosis is made: one type using reflected light (polychromatic plates, matching chips, 100-hue test or dichotomy test), the other using luminous signals (anomaloscope, spectroscope, lantern or threshold test).

This report summarizes a critical evaluation of the Ishihara test plates taken as a representative type of polychromatic plates used for the detection and classification of observers with defective color vision. (The effect of quality of illumination on the results will be reported separately.) With minor changes, this evaluation can be applied to other plates of the same type (Stilling's, Rabkin's and the recent American Optical Company's issue of selected plates for the use of the armed services). All have particular merits and defects, and even in different editions of the same test there are to be noted marked, and sometimes critical, variations due to changes in printing inks, printing papers, printing processes and age. The use of linseed oil bases for inks is hardly conducive to permanence of printed colors.

"Pseudo-isochromatic" means apparently or falsely equal colors; that is, the colors are trickily chosen to fall within the color diagram zones where the defective observers are most likely to be trapped. Since the apparent color of any object can be changed by modifying the light used to view it, all these plates must be viewed under special external conditions. The Ishihara plates are not particularly critical, and this is one of their defects. Such plates can be made very critical, but in so doing the effect of the illuminant is enhanced.

The Ishihara Test for Colour-Blindness was devised by Dr. Shinobu Ishihara, professor of ophthalmology, medical department, Imperial

From the Knapp Memorial Laboratories, Institute of Ophthalmology.

University of Tokyo, and was first described by him in 1917, when he was instructor at the military Medical Academy in Tokyo.[1] Since that time the test has run through eight or more Japanese editions, in the course of which some changes have been made in the colors used and in the number of plates in the series, although the basic principles have remained unaltered. The first edition (1917) we have been able to inspect through the courtesy of Dr. Arnold Knapp, professor emeritus of ophthalmology, Columbia University College of Physicians and Surgeons, New York, who has in his library a complimentary copy of this little known edition. In this edition the introduction is less complete than in the later editions; the red-purple pigment is bluer than was used later, and plates 8 and 9 consist of an entirely different color combination. These plates as first published showed the digits composed of small blue-green disks on a background of small yellow and violet disks. In all later editions the digits are composed of green disks and the background of yellow and red-purple disks. The second edition was copyrighted in 1920,[2] and later editions, through the sixth, are close copies of this. A third edition was published in 1922 and a fourth in 1924. These were distributed in this country by E. B. Meyrowitz, Inc., of New York. The first publication on the use of the Ishihara test in this country was in 1924, by Clark,[3] who gave us the information that the original plates were destroyed in the Japanese earthquake of 1923. A fifth edition appeared in 1925, at which time C. H. Stoelting Co., of Chicago, was made the sole distributing agent in the United States, and a sixth edition in 1932. These two editions became widely known and used in this country. Like the earlier editions, they consist of 16 plates, the first plate for demonstration and the detection of color malingering, the 12 succeeding plates for the detection of congenital color blindness of the red-green type and of monochromasy in literates and the last 3 plates for the detection of this defect in illiterates. A seventh edition appeared in 1936, an eighth in 1939 and a ninth in 1940.[4] These three editions contain 32 plates, the original 16 and 16 new plates of the same type but carrying different patterns. There are also some change in the pigments and a more liberal use of the two digit plate. The ninth edition was reprinted in London in 1943.

The fifth, seventh and ninth (British reprint) were the only editions available to us for experimental work and were, therefore, the ones used in the present study, and in a study to be reported separately, for a comparison of their value in detecting and analyzing deficiency in color vision.

In the words of the author of the test series:

> . . . this series of plates is intended to discover quickly and accurately congenital colour-blindness, the most common form of colour-blindness. . . . Congenital colour-blindness is of two kinds; total colour-blindness and red-green blindness, and these have each two forms; complete and incomplete. . . . Incomplete total colour-blindness is the result of deficiency in the red-green sense and an accompanying weakness of the blue-yellow sense.[5]

Ishihara further classifies red-green blindness into red blindness (the type called by von Kries protanopia) and green blindness (called by von Kries[6] deuteranopia). Ishihara designates red blindness and green blindness also as complete and incomplete but does not describe what he means by this designation. He makes no claim to detecting the degree or extent of the color defect; i. e., a qualitative but not a quantitative evaluation is indicated. However, no critical score is given to indicate the dividing line between poor but normal color vision and defective color vision.

In constructing his test plates, Ishihara was obviously influenced by Stilling's Pseudo-Isochromatic Plates for Testing the Color Sense, but no plates are a direct copy and certain innovations are made.

The plates intended for testing color-blind literates consist of six series, 2 plates of each series in the fifth edition and 4 of each series in the seventh and ninth editions.

In series 1, which might be called a "transformation pattern" series, a digit composed in general of blue-red and yellow-red disks, varying from 1 to 5 mm. in diameter, is seen by the subject with normal color vision on a background composed of yellow-green and blue-green disks of these sizes, the disks of both digit and background varying as to chroma and value. For the subject with defective red-green vision, the yellow-green

1. Ishihara, S.: Tests for Colour-Blindness, Handaya, Tokyo, Hongo Harukicho, 1917.

2. Dr. Janet Howell Clark, professor of biophysics and dean of women of the University of Rochester, and Dr. Anna M. Baetjer, associate in physiological hygiene, School of Hygiene and Public Health, Johns Hopkins University, gave us access to this edition. The book is a complimentary copy belonging to the late Dr. W. H. Howell, former professor of physiology and director of the School of Hygiene and Public Health, Johns Hopkins University.

3. Clark, J. H.: The Ishihara Test for Color Blindness, Am. J. Physiol. Optics **5**:269-276 (July) 1924.

4. Mr. R. Koller, of C. H. Stoelting Co., Chicago, gave us access to the sixth, eighth and ninth (Japanese) editions.

5. Ishihara,[1] ed. 5, Tokyo, Kanehara, 1925.

6. von Kries, J.: Ueber Farbensysteme, Ztschr. f. Psychol. u. Physiol. d. Sinnesorgane **13**:241-324, 1897.

disks of the background and the yellow-red disks of the digit are indistinguishable, and a pattern is formed of the blue-red and blue-green disks which appear to him as a bluish digit on a yellowish background. The pattern is thus transformed for the subject with defective color vision into a different digit from that seen by the subject with normal color vision; e. g., from an 8 to a 3. This series, as presented in the fifth edition, traps deuteranopes only.

Series 2 also is a transformation series, but with the colors of background and pattern reversed, that is, blue-green and yellow-green disks form the digit on a background of yellow-red and blue-red disks. As in series 1, the subject with defective color vision sees a bluish digit on a yellowish background. All subjects with defective color vision fail this series when tested with the fifth and seventh editions.

Series 3 and 4 are of the "vanishing digit" type, used by Stilling. In series 3 the digit is formed of yellow-red disks on a background of yellow-green and blue, and in series 4, of blue-green disks on a background of blue-red and yellow. These digits are plain to subjects with normal color vision but are rarely seen by subjects with defective color vision unless the defect is slight. This series fails to trap many deuteranomalous subjects and observers with low color discrimination.

Series 5 contains a "hidden digit," which supposedly is seen only by persons with defective color vision but is too often quite visible to subjects with normal color vision as well. The series has little diagnostic value.

Series 6 consists of two digits (e. g., 26), one in red and one in red-purple disks on a background of gray disks of different brightness. Protanopes, whose neutral region is in the red, are supposed to see only the red-purple digit (the 6), and deuteranopes, whose neutral region is in the red-purple, only the red digit (the 2). This series helps to differentiate dichromasy from trichromasy and is adequate to classify only deuteranopes as to type of defect.

The remaining plates are for testing illiterates, who are instructed to follow "with their eye" a winding line between two terminal x's. In some of the plates this is an easy task for subjects with normal color vision but is difficult for subjects with defective color vision; in others the path is readily visible to subjects with defective color vision but is not plain to persons with normal color vision. These plates are difficult to administer and score, and, since only literates were included among the subjects used in the present study, they will not be considered further.

EVALUATION OF THE FIFTH EDITION OF THE ISHIHARA TEST AS A MEANS OF DETECTING AND CLASSIFYING DEFECTIVE COLOR VISION

The Ishihara test (fifth edition) was included as one test in a battery used in the Knapp Memorial Laboratories to study the persons with defective color vision referred to the laboratories. These subjects were not selected on the basis of any statistical survey. They included patients referred to us by ophthalmologists, men who had been rejected by the armed forces on the basis of defect in color vision and subjects obtained from some of the New York city high schools, from the Vanderbilt Clinic and from other sources. The battery included the following tests, which have been described in the literature:

Ishihara Tests for Colour-Blindness (fifth edition).[5]

Rabkin Polychromatic Plates for Testing Colour Vision (second edition).[7]

Pseudo-Isochromatic Plates for Testing Color Perception, engraved and printed by Beck Engraving Co., Inc., and published and distributed by the American Optical Company.[8]

Farnsworth-Munsell 100-Hue Test.[9]

Farnsworth Dichotomous Test.[9]

Inter-Society Color Council Single Judgment Test for Red-Green Discrimination.[10]

Nagel's anomaloscope.[11]

Several additional tests were used which have not as yet been described in print, notably a series of polychromatic plates and a new form of anomaloscope devised in this laboratory. The results of the entire battery of tests aided in the classification of the type and extent of the defects in color vision. Our data thus permit us to evaluate each of the aforementioned tests as a diagnostic or screening medium and as a medium for the differential classification of the type and extent of the defect.[12] The present

7. Rabkin, E.: Polychromatic Plates for Testing Colour Vision, ed. 2, Kharkov, State Medical Publishing House of the USSR, 1939.

8. American Optical Company: Pseudo-Isochromatic Plates for Testing Color Perception, Philadelphia and New York, Beck Engraving Co., 1940.

9. Farnsworth, D.: Farnsworth-Munsell 100-Hue and Dichotomous Tests, J. Optic. Soc. America **33**:568-578 (Oct.) 1943.

10. Hardy, L. H.: Single Judgment Test for Red-Green Discrimination, J. Optic. Soc. America **33**:512-514 (Sept.) 1943.

11. Nagel, W. A.: Zwei Apparate für die augenärztliche Funktionsprüfung. Adaptometer und kleines Spektralphotometer (Anomaloskop), Ztschr. f. Augenh. **17**:201-222, 1907.

12. A test for color blindness may be a simple diagnostic or screening medium, in which case the purpose

(Footnote continued on next page)

paper, however, will be confined to the work with the Ishihara test, mention being made of the battery of tests because of its use in the classification of the subjects as to type and extent of defect. Because this battery of tests was so comprehensive, it is understandable why we chose for inclusion the fifth edition of the Ishihara test, consisting of 16 plates, rather than one of the longer editions. The comparison of results given by the various editions at our disposal was made on a smaller group of subjects, including some with each type of defective color vision. This work is reported separately.

The entire battery of tests was given to 106 subjects: 23 deuteranomalous and 12 protanomalous subjects (anomalous trichromats); 15 deuteranopes and 17 protanopes (dichromats); 7 persons showing generally low color discrimination sufficient to amount to a defect in color vision, and 32 persons who showed low color discrimination to a lesser degree which would not be sufficient to amount, in all probability, to a real defect in color vision. This last group we have classified as "low normal." This classification of subjects follows the scheme outlined by two of us (L. H. H. and G. R.).[13] For the classifying of anomalous trichromasy particular reliance had to be placed on the results with the anomaloscope, our special polychromatic plates and the Farnsworth Dichotomous Test; for the classifying of low color discrimination and low normal color vision on these tests and, in addition, on the Farnsworth-Munsell 100-Hue Test.[14]

For all work with pigment test material a close approximation to I.C.I. Illuminant C was used. (Illuminant C approximates a color tem-

is merely to screen out the subjects with defective color vision from those with normal color vision; or it may be a differential classifying medium, in which case it is designed to determine both the type and the extent of the defect. It is our belief that the ideal tests for color blindness should serve both functions. 1. For some purposes the simple screening test is adequate, and its simplicity of operation and interpretation is an advantage. 2. For such purposes as industrial employment, vocational guidance and other means of adapting the environment of the person with defective color vision so that it may best serve his practical and esthetic needs, it is important to know how much defect is present and how color stimuli appear to him. There are many vocations, such as automotive wiring, which are open to anomalous trichromats but which cannot be handled by dichromats. Even in this field distinctions must be made. For example, the anomalous trichromat who can distinguish the difference in color between new-coded wires viewed under good illumination is able to work on new motors; but he cannot distinguish this difference under flashlight illumination or when the wires have become soiled by use and is, therefore, unable to do repair work on the motors. It is often necessary, also to know the type of defect and whether or not there is distortion of the brightness value of color stimuli. It would seem that a deuteranope, who has little, if any, distortion of the brightness aspect of the colors, would be useful in certain types of heterochromatic photometry, while the protanope, who sees the long wave end of the spectrum as darker and the short wave end as lighter than normal, could not do this work. Again, a deuteranope is less likely to distinguish between the customary red, green and amber traffic signals than the protanope, who sees the green signal as bright and the red one as dim. On the other hand, the protanope may find it difficult to see a red traffic signal during night driving.

13. Hardy, L. H., and Rand, G.: Recent Developments in Color Vision Testing, Graduate Lecture, American Academy of Ophthalmology Outlines of Instructional Courses, Continuous Course No. 6 and 7, 1944. Hardy, L. H.; Rand, G., and Rittler, M. C.: Color Vision and Recent Developments in Color Vision Testing, Arch. Ophth., to be published.

14. To persons not familiar with the classic terminology of color blindness and those to whom our article [13] is not available, the following simple explanation of terms may be of value.

In the Young-Helmholtz theory there are three factors in the color vision process: (1) the red primary, proto, or first, process; (2) the green primary, deutero, or second, process, and (3) the blue primary, trito, or third, process. Hence one would expect to find three types of color vision: (1) trichromatic, in which all three processes function; (2) dichromatic, in which only two processes function, and (3) monochromatic, in which there is only one differentiated physiologic process. Trichromatic color vision means that all three processes are functioning, but since one or more of the processes may function aberrantly, there will be as a result three types of trichromatic color vision: (1) normal; (2) anomalous, in which one or more of the processes is weak, and (3) low discrimination, in which apparently all processes are weak. Anomalous trichromasy is designated according to the process which is weak as (1) protanomaly, or predominantly red weak; (2) deuteranomaly, or predominantly green weak, and (3) tritanomaly, or predominantly blue-yellow weak. Dichromatic color vision means that only two of the processes are functioning and hence is similarly divided into three types: (1) protanopia, formerly called red blindness; (2) deuteranopia, formerly called green blindness, and (3) tritanopia, formerly called blue-yellow blindness. As a mnemonic, we might point out the recurrent triad: There are three kinds of abnormal color vision: (1) anomalous trichromasy (three types—protanomaly, deuteranomaly and tritanomaly); (2) dichromasy (also three types—protanopia, deuteranopia and tritanopia), and (3) monochromasy. Protanopia exhibits a shortened red end of the spectrum, a neutral area at about 493 millimicrons (in the blue-green), a second neutral band in the red (at 493 c) and a brightness peak, which is shifted toward the violet. Deuteranopia shows a neutral region in the green (at about 497 millimicrons), a second neutral band in the red-purple (at 497 c) and no shift in the luminosity peak. Tritanopia shows a shortened blue end of the spectrum and a neutral band in the yellow-green. The luminosity peak is not shifted. These types, as well as corresponding anomalous trichromatic types, are best detected and classified by anomaloscopic tests.

perature of 6750° K. and represents average overcast skylight or average daylight.) This was obtained from the combination of a 100 watt gas-filled tungsten filament lamp (color temperature 2848° K.) and a Macbeth daylight glass filter, the combination giving a color temperature of 6700° K.[15]

The illumination on the test material was about 25 foot candles. The testing distance in the case of polychromatic test plates was about 30 inches (76 cm.). Responses should be immediate (within two seconds). Hesitant or studied responses are viewed with suspicion.

The results for the Ishihara test, fifth edition, are summarized in tables 1 to 4. They will be discussed under three headings: (1) analysis of the individual plates; (2) significance of performance score on the entire test; (3) differential classification of deuteranopia and protanopia, deuteranomaly and protanomaly.

TABLE 1.—*Analysis of Individual Plates in the Ishihara Test, Fifth Edition*

Type of Defective Color Vision	Number of Subjects	Percentage of Subjects with Each Type Passing Individual Plates											
		Series No.											
		1		2		3		4		5		6	
		Plate No.		Plate No.		Plate No.		Plate No.		Plate No.		Plate No.	
		2	3	4	5	6	7	8	9	10	11	12	13
Anomalous Trichromasy													
Deuteranomaly	23	43	43	0	0	13	22	17	39	13	0	17	30
Protanomaly	12	67	67	0	0	0	0	8	8	0	0	8	17
Dichromasy													
Deuteranopia	15	0	7	0	0	0	0	0	0	0	0	0	0
Protanopia	17	53	59	0	0	0	0	0	0	24	18	0	0
Low discrimination	7	57	71	0	0	14	57	14	43	0	0	43	86
All types of defective color vision	74	42	46	0	0	5	12	8	18	9	4	11	20
Low normal color vision	32	100	100	100	78	87	100	100	94	87	84	97	97

Analysis of the Individual Plates.—In table 1 is shown for the subjects with each type of defective color vision and for the subjects with low normal color vision the percentages who pass the individual Ishihara plates, designated by both series number and plate number. The first five horizontal rows of figures show these percentages for each type of defective color vision and demonstrate the value of the individual plates for detecting each type. The lower two rows present for comparison the percentages for the subjects with defective color vision as a group and for the subjects with low normal color vision as a group. This comparison shows the value of each plate as a diagnostic or screening medium.

In this table it is seen that the plates of series 2 are the only ones which were failed by all the subjects with defective color vision. Plate 4 of this series was passed by all the subjects with normal color vision, and plate 5 by only 78 per cent of this group. Plate 4 is therefore unique in that all the subjects with defective color vision fail it and all the subjects with normal color vision pass it. Judged by our observers, then, this plate when properly administered furnishes an ideal screening or diagnostic test; used with Mazda light, as will be shown separately, its efficiency for detection of deuteranomalous subjects drops from 100 to 43 per cent. The remaining plates are much less successful. For example, the plates of series 3 serve to detect deuteranopes, protanopes and protanomalous subjects but not all deuteranomalous subjects or observers with low color discrimination; the plates of series 4 and 6 detect deuteranopes and protanopes but not all deuteranomalous and protanomalous observers or subjects with low discrimination; the plates of series 5 detect deuteranopes and protanomalous subjects and observers with low discrimination but not all deuteranomalous subjects or protanopes. The plates of series 1 are the least effective, except for deuteranopes. They are particularly ineffective for protanopes and protanomalous subjects who tend to see the digit (which is composed of dominantly reddish disks) as a dark figure and the background (which is composed of greenish disks) as light. For this reason more than 50 per cent of protanopes and 67 per cent of protanomalous subjects pass these plates. Protanopes pass the plates of series 5, too, in a higher percentage than would be expected; that is, they fail to see the "hidden digit." It is because of their

15. The Macbeth Daylighting Company has designed an excellent table lamp and book rest to provide convenient, standard conditions for tests of the polychromatic plate type. The color temperature of the illumination supplied is 6800° K; the amount of illumination on the test material is about 40 foot candles; the illumination is even and falls on the test material at an angle of approximately 45 degrees, and the test material is supported so that it is viewed at an angle of 90 degrees. The complete unit, which is called the Macbeth Easel Lamp, provides ideal lighting conditions for the administration of color tests of this type. It is supplied by the Macbeth Daylighting Company, 227 West Seventeenth Street, New York.

success with the plates of series 1 and 5 that protanopes achieve, on the average, a higher performance score on the entire test than deuteranopes and nearly the same score as protanomalous subjects (table 2).

Significance of Performance Scores on Entire Test.—Table 2 shows the performance scores obtained on the entire Ishihara test. In this table are given for each type of defective color vision and for low normal vision (*a*) the average score achieved by each group, (*b*) the median, or middle, score for each group and (*c*) the range of scores, from lowest to highest, achieved by the subjects within each group. A score of 100 indicates that correct responses were given to all 12 plates; a score of 0, that no correct responses were given, and scores between 0 and 100 indicate the percentage of the 12 plates to which correct responses were given. As in table 1, the first five horizontal rows of figures give the data for the subjects with each type of defective color vision and the lower two rows, the data for the subjects with defective color vision as a group and for the subjects with low normal color vision as a group.

TABLE 2.—*Significance of Performance Score* *

Type of Defective Color Vision	No. of Subjects	Average Score	Median Score	Range of Scores
Anomalous Trichromasy				
Deuteranomaly	23	20	17	0- 58
Protanomaly	12	15	17	0- 33
Dichromasy				
Deuteranopia	15	1	0	0- 8
Protanopia	17	13	17	0- 33
Low discrimination	7	31	25	8- 58
All types of defective color vision	74	15	13	0- 58
Low normal color vision	32	94	100	75-100

* The performance score is the percentage of the 12 plates to which correct responses were given.

In table 2 it is seen that (*a*) the subjects with defective color vision as a group have an average performance score of 15 and the subjects with low normal vision a score of 94; (*b*) deuteranomalous subjects have an average score of 20 and deuteranopes an average score of 1, and (*c*) protanomalous subjects have an average score of 17 and protanopes an average score of 15. On the average, then, on the basis of the performance scores, the Ishihara test would seem to separate persons with defective color vision from those with normal color vision and deuteranomalous subjects from deuteranopes. However, for the purpose of screening or of classification, it is not the average score achieved by a group that is important but the score achieved by the individual subject. This is shown in the last column of table 2, in which for each type of defective color vision is given the range of scores, from lowest to highest, achieved by the subjects having that type of defect. This column shows clearly the overlapping of scores between the groups with the various types of defective color vision and thus demonstrates the impossibility of classifying the type of color defect on the basis of score alone. There is, however, no overlapping of scores between the group of subjects with defective color vision taken as a whole and the group of subjects with low normal color vision, the lowest score of the latter group being 75 and the highest score of the former being 58. In round numbers, then, it would seem that a critical score of 60 might be accepted as the dividing line between the performance of subjects with defective color vision and normal performance on the Ishihara test.

The overlapping of scores between groups with the various types of defective color vision is brought out still more clearly in table 3, which shows the distribution of performance scores for each type. For representation in this table, the following classification of performance scores was made: 0 (failure on all plates); 8 to 17 (passing 1 or 2 plates); 25 to 42 (passing 3 to 5 plates); 50 to 67 (passing 6 to 8 plates); 75 to 92 (passing 9 to 11 plates), and 100 (passing all plates). The overlapping of scores between the groups with the various types of defective color vision is clearly shown in this table, and consequently the inability of the Ishihara test to yield a classification of either type or extent of defect on the basis of performance score.

TABLE 3.—*Distribution of Performance Scores on the Ishihara Test (Fifth Edition)*

Type of Defective Color Vision	No. of Subjects	Number of Plates Passed: Percentage of Each Group Achieving Scores of: 0	8-17	25-42	50-67	75-92	100
Anomalous Trichromasy							
Deuteranomaly	23	30	35	22	13	0	0
Protanomaly	12	25	50	25	0	0	0
Dichromasy							
Deuteranopia	15	93	7	0	0	0	0
Protenopia	17	18	76	6	0	0	0
Low discrimination	7	0	29	43	29	0	0
All types of defective color vision	74	36	41	14	9	0	0
Low normal color vision *	32	0	0	0	0	47	53

* In this group, 53 per cent passed all 12 plates (score 100); 22 per cent passed 11 plates (score 92); 22 per cent passed 10 plates (score 83), and 3 per cent passed 9 plates (score 75).

The following points are brought out in tables 2 and 3:

1. The scores of deuteranomalous and protanomalous subjects and of observers with low

color discrimination have a wider range of scatter than those of deuteranopes and protanopes. That is, as seems probable, their defect may vary from a slight to an extreme anomaly or low discrimination, while dichromats having a defect in red-green vision would present a more homogeneous group, at least when studied by means of tests designed primarily to detect defects in red-green vision.

2. All persons with defective color vision of the red-green types, as well as persons having low color discrimination amounting to a defect, are screened from the group having normal and low normal color vision if 60 is taken as the critical score on the Ishihara test (fifth edition). In the group tested no subject with defective color vision achieved a score of more than 58, and no subject with low normal color vision had a score of less than 75.

adequate to separate subjects with defective color vision from those with normal color vision when the test is properly administered, but it is not adequate to make the equally important differential classification of type and extent of defect. It remains to discuss the type of response to certain plates as a clue to this classification.

In the Ishihara test the plates of series 6 are the only ones designed for this purpose. On these plates a double digit is presented (26 on plate 12 and 42 on plate 13). On each plate the left hand digit is composed of red disks, the right hand digit of red-purple disks and the background of gray disks. According to Ishihara, the subject with deuteranopic type of red-green blindness will be able to read only the red digit, and the protanopic type, only the red-purple digit. Our data have been analyzed to determine the effectiveness of these plates for making this differential classification.

TABLE 4.—*Analysis of Value of Plates 12 and 13 (Series 6) as a Means of Differentiating Deuteranopia and Protanopia, and Deuteranomaly and Protanomaly*

Type of Defective Color Vision	Number of Subjects	Percentage of Subjects Correctly Classified by				Percentage of Subjects Incorrectly Classified by				Percentage of Subjects Not Classified by			
		Plate		Both Plates	Either Plate	Plate		Both Plates	Either Plate	Plate		Both Plates	Either Plate
		12	13			12	13			12	13		
Anomalous Trichromasy													
Deuteranomaly	23	65	65	57	74	0	0	0	0	35	35	43	26
Protanomaly	12	50	83	50	83	8	0	0	8	42	17	50	8
Dichromasy													
Deuteranopia	15	93	93	87	100	0	0	0	0	7	7	13	0
Protanopia	17	41	59	29	71	6	6	6	6	53	35	65	23

3. No cleancut separation as to extent or type of defect can be based on performance scores. While in general the dichromats had lower scores than the anomalous trichromats, a majority of the latter group (65 per cent of deuteranomalous and 75 per cent of protanomalous subjects) had scores between 0 and 17, the range which includes 100 per cent of the deuteranopes and 94 per cent of the protanopes tested. Scores cannot, therefore, be used to indicate either the extent or the type of the defect.

4. Of the deuteranopes tested, 93 per cent failed all the plates in the Ishihara test; of the protanopes, only 18 per cent failed them all. The fifth edition of this test, then, "traps" deuteranopes more decisively than persons with the other types of defective red-green vision.

Differential Classification of Deuteranopia and Protanopia, and Deuteranomaly and Protanomaly.—So far we have discussed performance scores on the Ishihara test both as a means of detecting the presence of a defect in color vision and as a means of classifying the type of defect, and we have shown that the score achieved is

Difficulty is encountered when other types of response are given, such as the correct reading of both digits or of neither (in which cases, of course, no interpretation is possible) or the incorrect reading of one or both digits. In scoring the responses to these plates, the following plan was adopted: A subject was called "classified" as to type of defect by the plate in question when one digit was read correctly and the other was not seen at all or was read incorrectly. When both digits were read correctly, when neither was read correctly or when one was read incorrectly and the other was not seen at all, the subject was called "not classified." In short, "a hit and a miss" are necessary in order to utilize this plate as a test for differential classification. The results obtained are shown in table 4.

The following points are brought out in table 4:

1. The plates of series 6 are better designed to classify deuteranopes as to type of defective red-green vision than deuteranomalous observers. The failure in case of the latter group is due either to an incorrect reading of the significant digit or to a correct response to both digits.

2. The plates of series 6 are better designed to classify protanomalous observers as to type of defective color vision than protanopes. The failure in the latter group is due either to an incorrect reading of the significant digit or to inability to see any digit.

3. Plates 12 and 13 give equally good results for the deuteranopic type, but plate 13 is better than plate 12 for distinguishing the protanopic type (83 and 59 per cent with plate 13, as compared with 50 and 41 per cent for plate 12).

4. One protanomalous observer and 1 protanope of our group would have been classified as deuteranopes by one or both plates of series 6; with Mazda light this number would have been greatly increased. None of the deuteranopic type were wrongly classified.

5. It is seen, for example, that whereas 41 per cent of the protanopes were correctly typed ("a hit and a miss") by plate 12, 59 per cent of this group were correctly typed ("a hit and a miss") by plate 13. However, if the indicated response was required from each plate (i. e., "a hit and a miss" on each plate), only 29 per cent of the protanopes were correctly typed. On the other hand, if the indicated response was required from only one of the two plates, and the results obtained from the other ignored, then 71 per cent of the protanopes were correctly typed, the second plate yielding an ambiguous response.

CONCLUSIONS

The following conclusions concerning the fifth edition of the Ishihara test seem justified:

1. Properly administered, the test affords a good rough device for screening persons with defective red-green vision from persons with normal color vision if a performance score of 60 is taken as the critical score. In the group tested, no subject with defective color vision scored more than 58 and no subject with low normal color vision less than 75.

2. No analysis as to type or extent of defect can be based on performance scores.

3. The test yields a lower score for the deuteranopic than for the protanopic type of defective color vision. (This is, in effect, a "weighting," which is lost with more yellowish illumination.)

4. The plates of series 6, which were designed to distinguish between deuteranopes and protanopes, are adequate to classify deuteranopes alone, when the report on either of the two plates is taken as indicative. On this basis, only 74 per cent of the deuteranomalous subjects, 83 per cent of the protanomalous observers and 71 per cent of the protanopes were correctly typed.

5. The Ishihara test is a gross test for defective red-green vision. It fails to classify the type of defective color vision (protanopia, protanomaly, deuteranopia, deuteranomaly, tritanopia, tritanomaly) and cannot be used to give a satisfactory evaluation of the extent or the degree of the defect, no matter how carefully administered.

6. Plate 4 is a practically perfect screening test when properly used; but if used with Mazda light, its efficiency drops from 100 to 43 per cent for deuteranomalous subjects.

23 East Seventy-Ninth Street.

STEREOSCOPIC SENSITIVITY IN THE SPACE EIKONOMETER

KENNETH N. OGLE, PH.D., AND VINCENT J. ELLERBROCK, M.S.
HANOVER, N. H.

Recent experimental work has shown that the space eikonometer is a suitable instrument for the measurement of aniseikonic errors.[1] This instrument [2] differs from the standard eikonometer in that the measurement depends on binocular (stereoscopic) perception rather than on a direct comparison of image sizes. In the space eikonometer all empiric clues which might affect the apparent relative positions of selected test elements are eliminated, and their apparent orientation can be perceived only by binocular (stereoscopic) space perception. If an aniseikonic error exists between the two eyes, these test objects will appear incorrectly oriented to the subject, according to the nature of the error present. By means of suitable size lenses the relative sizes of the images in the two eyes can be changed until the test objects appear correctly oriented.[3] The magnifications introduced to achieve this measure the aniseikonic error.

To complete the study of the visual factors on which this instrument is based, the stereoscopic sensitivity for the particular test objects used under optimum conditions must be known. The purpose of this paper is to report the results of a series of rather comprehensive experiments in which these sensitivities were determined.

DESCRIPTION OF INSTRUMENT

Only a brief description of the space eikonometer need be repeated here. Complete descriptions and technics for operation can be found elsewhere.[4]

As shown in figure 1, the apparatus consists of three parts: (1) the arrangements for supporting and shielding the test elements, (2) a head support and (3) the optical test system.

The test elements consist of an oblique cross and four plumb lines. The cross is composed of two intersecting smooth red cotton cords stretched between the corners of a 5 foot (152 cm.) square wooden frame, which is set up vertically a distance of 3 meters from the eyes of the subject. The plane of the cross is set at right angles to and centered with respect to the objective median and horizontal planes of the subject. A plumb line (smooth white cord) can be suspended through the center and in the plane of the cross.

Two pairs of plumb lines (smooth cord) are set parallel to the plane of the cross, one pair (green) 60 cm. to the front and the other (white) a similar distance

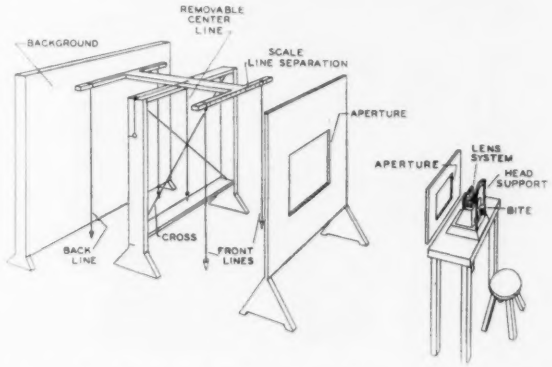


Fig. 1.—Perspective drawing of essential parts of apparatus for determining the sensitivity of the test elements of the space eikonometer.

to the rear. These cords are suspended from 2 meter sticks, which provide a means for locating the plumb cords at different separations.

The test elements were seen against a background of stretched black velvet cloth. In front of the elements, about 1 meter from the subject, was erected a screen (painted flat black), in which an aperture was cut. This aperture restricted the field of view to the elements and prevented their ends from being seen. The angular field of view was 21 degrees in the horizontal and 15 degrees in the vertical meridian.

A uniform illumination was provided by four 18 inch (45.5 cm.) (40 watt) Lumiline lamps arranged in a plane and equally spaced on each side of and above and below the center of the cross. With this illumination the test elements appeared to stand out vividly against the black background, without shadows. The brightness of the cords was about 0.6 millilamberts.

The entire space between background and screen was enclosed by black sateen cloth. The general illumination of the room, supplied indirectly by a 15 watt lamp, was low and diffuse.

From the Research Division of the Dartmouth Eye Institute, Dartmouth Medical School.

1. Ogle, K. N.: Association Between Aniseikonia and Anomalous Binocular Space Perception, Arch. Ophth. **30**:54-64 (July) 1943. Burian, H. M., and Ogle, K. N.: Meridional Aniseikonia at Oblique Axes, ibid. **33**:293-310 (April) 1945. Ogle, K. N., and Madigan, L. F.: Astigmatism at Oblique Axes and Binocular Stereoscopic Spatial Localization, ibid. **33**:116-127 (Feb.) 1945.

2. Ames, A., Jr.: The Space-Eikonometer Test for Aniseikonia, Am. J. Ophth. **28**:248, 1945.

3. Ogle, K. N.: Theory of the Space Eikonometer, Arch. Ophth., to be published.

4. Ogle and associates.[1] Ames.[2]

The head of the subject was held before the optical test system by forehead and temple rests and by a wax bite. This arrangement tends to eliminate head movements and depth perception clues from parallax.

To determine the stereoscopic sensitivity for the several test elements, the disparities between the images of these elements in the two eyes were modified and measured by an optical system before the eyes (fig. 2). The optical system used in the experiments reported here consisted of an over all and a meridional adjustable size unit [5] before the left eye and the right eye, respectively. The meridional unit could be rotated so as to magnify only in the horizontal meridian (axis 90) or in the vertical meridian (axis 180). By means of an attachment to the meridional unit its magnification could be set and measured in steps of 0.05 per cent with a maximum error of 0.02 per cent.[6]

A device for changing the declinations between the images of the two eyes was mounted in front of the adjustable units. This consisted of two matched 2 per

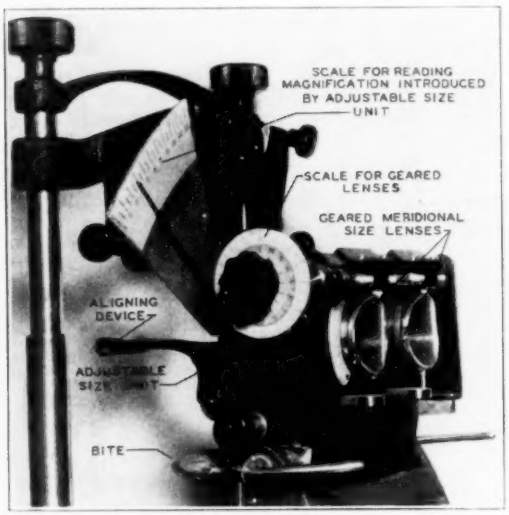

SCALE FOR READING MAGNIFICATION INTRODUCED BY ADJUSTABLE SIZE UNIT
SCALE FOR GEARED LENSES
GEARED MERIDIONAL SIZE LENSES
ALIGNING DEVICE
BITE

Fig. 2.—Photograph of the test lenses used in the study of the sensitivities of the space eikonometer.

cent meridional size lenses mounted in geared rings and so arranged that they could be rotated equally but in opposite directions when activated by a screw. A drum with a scale was attached to the screw, to read the amount of rotation of the geared lenses. These lenses could be set, without significant error, in steps of 1.25 degrees, introducing a change in vertical declination of 0.03 degree.

Before the right eye a shutter was placed between the adjustable size unit and the geared lenses. When lowered, this occluded the right eye except for a small region (about 1 minute of arc) seen through a hole in the center of the shutter. This hole permitted binocular fixation of the center of the cross, when binocular perception of the test elements was interrupted, and kept the eyes properly converged.

5. Ogle, K. N.: An Optical Unit for Obtaining Variable Magnification in Ophthalmic Use, J. Optic. Soc. America **32**:143-146 (March) 1942.

6. These values were determined on the so-called dioptoeikonometer (Ogle, K. N., and Ames, A., Jr.: Ophthalmic Lens Testing Instrument, J. Optic. Soc. America **33**:137-142 [March] 1943).

PROCEDURE AND METHOD

Three measurements are made on the space eikonometer which determine the aniseikonic error of a given subject. The image size difference in the horizontal meridian (axis 90) is determined by finding what magnification must be introduced before one eye in the horizontal meridian to cause the two front cords of the instrument to appear at the same distance from the subject. A method of limits is used. The image size difference in the vertical meridian (axis 180) is then determined by finding what magnification must be introduced before one eye in the vertical meridian in order to correct any apparent rotation of the cross about a vertical axis. The declination error [3] which is indicative of a meridional aniseikonic error at an oblique axis is determined by finding the angle through which the geared size lenses have to be turned to correct any apparent inclination of the cross about a horizontal axis. The purpose of this study is to obtain data on sensitivity for these three types of measurements.

The problem of determining the stereoscopic sensitivity of persons by finding a measure of the thresholds in such an experimental apparatus is primarily one of psychometrics. Several methods of determining these thresholds are generally recognized, but the so-called method of constant stimuli [7] is usually considered the most reliable. This method, as applied here, will be clear from the following discussion:

Consider the problem of finding a measure of the stereoscopic sensitivity (acuity) of a subject for the apparent displacements of the front vertical plumb lines of the space eikonometer. If one magnifies the image of the right eye by a given amount in the horizontal meridian, and thus changes the disparities between the retinal images of the two vertical lines in the two eyes, the right line will appear farther away from the subject than will the left line. If the size of the image is decreased in the horizontal meridian, the right cord will appear nearer than the left. The least change in magnification which will be recognized in an apparent displacement of the two cords would be a measure of the threshold and, therefore, of the sensitivity. Such a minimal change, however, will not always be recognized; and, again, sometimes even a smaller change will be seen, so that the problem becomes a statistical one. In what percentage of times will certain given changes in magnification be recognized in an apparent displacement of the two cords?

A few preliminary trials usually establish the approximate limits in change of magnification for which the right cord will first appear just farther away and then just nearer than the left cord. This range was then broken into five equally spaced changes in magnification, and each of these changes was presented to the subject 40 times in a random order.[8] Thus, a series of 200 judgments of random changes in magnification was obtained as to whether the right cord appeared farther or nearer than the left cord. So far as possible, the subject was asked to give either one or the other of

7. For a discussion of these methods see Guilford, J. P.: Psychometric Methods, New York, McGraw-Hill Book Company, Inc., 1936, p. 166.

8. The random order was obtained from Fisher, R. A., and Yates, F.: Statistical Tables for Biological, Agricultural and Medical Research, London, Oliver & Boyd, 1938, table 33, p. 82.

these responses, though occasionally a "can't tell" or a doubtful response was allowable.[9]

The shutter was lowered immediately after a response from the subject, and all changes in the adjustable size unit were made while the right eye could see only the small central part of the cross.

The tests were made under the same conditions as those observed when data are obtained with the space eikonometer. Consequently, no limit was placed on the time required by the subject to make a judgment, though on the average this varied between two and eight seconds. During this period the subject was also free to use ocular movements in making the judgment if he so desired. He was cautioned, however, to use the same criterion throughout a given experiment.

TABLE 1.—*Typical Set of Data Obtained for Determining a Measure of the Stereoscopic Sensitivity for the Front Cords on the Space Eikonometer* *

Magnification Setting, Percentage	Judgment Category	Number of Judgments in Each Category	Percentages †
0.90	Nearer	2	5.1
	Doubtful	1	
	Farther	37	94.9
0.85	Nearer	10	26.3
	Doubtful	2	
	Farther	28	73.7
0.80	Nearer	24	64.9
	Doubtful	3	
	Farther	13	35.1
0.75	Nearer	31	79.5
	Doubtful	1	
	Farther	8	20.5
0.70	Nearer	39	97.5
	Doubtful		
	Farther	1	2.5

* The number of judgments in which the right cord appears either "nearer" or "farther" than the left cord is shown as the magnifying unit before the right eye is changed in five different steps. The separation of the cords was 50 cm., corresponding to a visual angle of 12 degrees.

† The doubtful judgments were divided among the other two categories in proportion to the number of judgments in the "nearer" and the "farther" category.

The experiment was divided into two parts, of 100 judgments each, with a rest period of five to ten minutes between the parts. In any given experiment if the judgments were found difficult to make one minute rest periods were permitted after each 25 judgments, to reduce the possible effect of fatigue.

TREATMENT OF DATA

The data obtained in any experiment consisted of the number, and therefore the percentage, of the judgments for each magnification setting in which the right cord was judged to be "nearer" or "farther" than the left cord. These data can be studied graphically, a typical set of which is given in table 1 and illustrated in figure 3. The type of curve that describes these data is the well known psychometric curve.

That magnification setting for which 50 per cent of the judgments were "farther" and 50 per cent "nearer" indicates the most probable relationship between the relative size of the images in the two eyes for which the two front cords appeared at the same distance. At this setting of the test lenses the judgments were made by pure guesswork, and the distribution found was due solely to chance. At the extremes of the curve, there is nearly 100 per cent certainty that the apparent displacements of the cords due to the change in magnification from the 50-50 per cent point would be seen. Between these limits the proportion of judgments varies from near certainty to pure chance. From this curve the threshold of stereoscopic sensitivity can be read off directly, the value depending on what percentage of certainty one chooses to consider as the threshold point.

The entire psychometric curve for a given set of data can be described quantitatively by a single constant,[10] σ, the so-called standard deviation. This quantity is therefore generally accepted as a measure of the accuracy of any series of responses. Roughly, it is only slightly smaller than the average deviation of a series of settings if the subject were to adjust the magnification units. The smaller σ becomes, the steeper is the central part of the curve and the smaller is the threshold value. A small σ would then be associated with a high sensitivity; a large σ, with a low sensitivity. A change in magnification of 0.6745 σ would be recognized 50 per cent of the time.

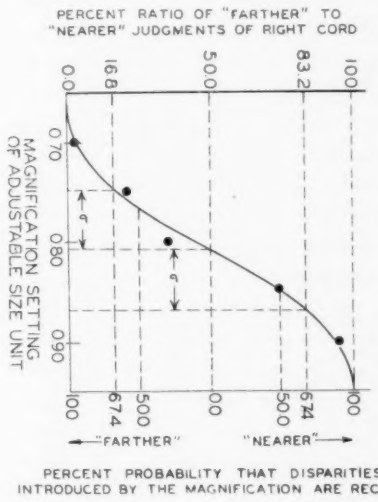


Fig. 3.—Typical psychometric curve obtained in determining the stereoscopic sensitivity for the front vertical cords of the space eikonometer to changes in the magnification of the image of the right eye in the horizontal meridian.

For a given set of data, the most probable value of σ and the value of that magnification at the center (the median) of the curve are found by standard statistical methods.[11]

This same method of constant stimuli was also used for studying the sensitivity to differences in size between the images in the two eyes in the vertical meridian

9. Here, a three category experiment was used. The number of doubtful responses was small, and for each stimulus these were distributed in the "nearer" and the "farther" category in proportion to the number of judgments in those categories. This procedure is today considered the proper way to treat doubtful responses (Guilford [8]).

10. The curve is the integral of the probability function, expressed by the equation $y = [1/\sigma \sqrt{2\pi}] \exp(-x^2/2\sigma^2)$.

11. The method of least squares is used, after attaching weights to the data according to the Müller-Urban process (Woodworth, R. S.: Experimental Psychology, New York, Henry Holt & Company, Inc., 1938, p. 408-419).

(axis 180), as judged by an apparent rotation of the cross about a vertical axis, and also for studying the sensitivity to changes in the vertical declination between the two eyes introduced by meridional size lenses at oblique axes, as judged by an apparent inclination of the cross about a horizontal axis.

Subjects.—Three subjects took part in the entire series of experiments. The ocular characteristics (for distant vision) of these subjects were as follows:

	Uncorrected Visual Acuity
V. J. E.	R. E. 20/15
	L. E. 20/15
N. W.	R. E. 20/15
	L. E. 20/15
K. N. O.	R. E. 20/15+
	L. E. 20/15

Two of the subjects (V. J. E. and K. N. O.) had had considerable training and experience with experiments requiring stereoscopic judgments. One subject (N. W.) had had no previous experience whatever in stereoscopic judgments.

RESULTS

A. Stereoscopic Sensitivity to Image Size Differences in the Horizontal Meridian (Axis 90).—The stereoscopic sensitivity to changes in the relative sizes of the image in the two eyes in the horizontal meridian as observed in an apparent displacement of the two front (green) vertical plumb lines of the space eikonometer was determined, first, as affected by changes in separation of the lines, and, second, as affected by the other configurations in the field of view.

It will be clear that, except for small separations, the images of one of the lines will always fall on the peripheral part of the retina of both eyes, whether the eyes are fixed on one or on the other line or in between the two lines. Now, steroscopic acuity in the last analysis cannot be better than the visual acuity for the same type of test objects. The visual acuity of the eye decreases rapidly from the center of the fovea toward the periphery, rapidly to about 10 degrees and then at a slower rate from there to 60 degrees. While the visual acuity decreases with the peripheral angle, the disparity between the images in the two eyes introduced by magnifying the image in one eye will increase with the peripheral angle (being proportional to that angle).[12] There was the possibility, therefore, that at some separation of the plumb lines a maximum sensitivity to changes in magnification might be found.

Table 2 gives the results obtained for different separations of the lines when the oblique cross was in the field of view and, again, when the cross was omitted and the front green cords were used alone. These data are also shown graphically in figure 4, in which the abscissas represent the separation of the cords and the ordinates the standard deviation in percentage magnification of the image of the right eye in the horizontal meridian.

Refractive Error	Phoria at 6 Meters	P. D., Mm.
+0.12 D. cyl., ax. 160	1½△ Exo.	62
+0.12 D. cyl., ax. 180		
+0.25 D. sph.	¾△ Exo.	60
+0.25 D. sph.	½△ RH	
+0.25 D. sph. ≅ −0.25 D. cyl., ax. 25	½△ Eso.	64
+0.25 D. sph. ≅ −0.12 D. cyl., ax. 10	¼△ RH	

An inspection of the table shows that, except for subject N. W., there was no significant difference when the cords were seen alone or when the other configurations, such as the cross, were included in the field of view. However, for subject N. W. the difference was significant, for repeated tests gave essentially the same results.

For the cords alone, the values of σ found for all 3 subjects were essentially in agreement. For separations of the lines from 30 to 60 cm. the sensitivities were essentially constant and maximum, the values for σ averaging about 0.055 per cent. For an average separation of 40 cm., this value of σ corresponded to an angular disparity between the two eyes of 18.7 seconds of arc.[13]

These results show that there is no critical separation of the cords for measurement of the size of the image at axis 90 so long as their separations are between 7 and 14 degrees.

TABLE 2.—*Tabulation of Data Which Indicate Stereoscopic Sensitivities for Changes in the Relative Size of the Images in the Two Eyes in the Horizontal Meridian (Axis 90) as Perceived in an Apparent Displacement of the Vertical Plumb Lines of the Space Eikonometer, for Various Separations of the Lines* *

Separation, Visual Angle	Standard Deviations, Percentage: With Cross V. J. E.	With Cross N. W.	With Cross K. N. O.	Without Cross V. J. E.	Without Cross N. W.	Without Cross K. N. O.
5 cm. (1.2°).....	0.130	0.147	0.153	0.118	0.124	0.128
					0.105	
					0.081	
15 cm. (3.6°).....	0.075	0.087	.....	0.079	0.078	.....
25 cm. (6.0°).....	0.065	0.054	0.069	0.051	0.053	0.080
				0.063		
50 cm. (11.9°)†...	0.056	0.073	0.056	0.061	0.051	0.059
		0.076			0.052	
					0.054	
65 cm. (15.2°)....	0.064	0.113	0.054	0.069	0.057	

* Values are the standard deviations (σ) in percentages of image size difference. The visual distance was 2.4 meters.

† This is the separation usually used on the space eikonometer.

12. Ames, A., Jr., and Ogle, K. N.: Size and Shape of Ocular Images: III. Visual Sensitivity to Differences in the Relative Size of the Ocular Images in the Two Eyes, Arch. Ophth. **7**:904-924 (June) 1932.

13. The relation between these data and visual acuity will be discussed in another paper.

B. Sensitivity to Image Size Differences in the Vertical Meridian (Axis 180).—Changes in the size of the image in one eye in the vertical meridian can be observed in the space eikonometer by an apparent rotation of the cross about a vertical axis. The right side of the cross appears nearer if the image of the right eye is increased vertically (axis 180). The data were obtained by the method of constant stimuli, the changes in magnification being made with the meridional adjustable size unit before the right eye.

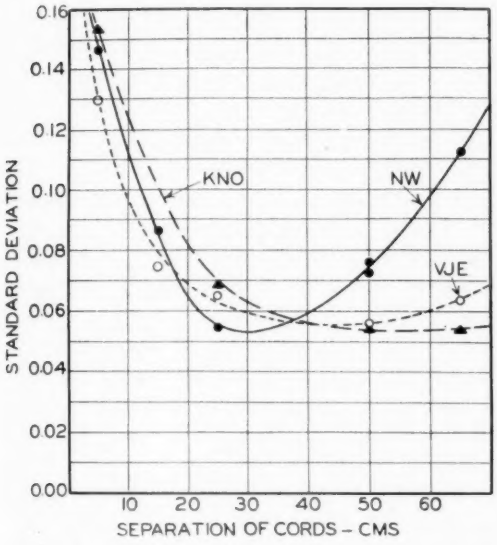


Fig. 4.—Data for the standard deviations indicating the stereoscopic sensitivities of 3 subjects to changes in the relative magnification of images in the two eyes of the vertical lines of the space eikonometer, for various separations of those lines.

The essential problem, in addition to determining the sensitivity to the cross as used in the space eikonometer, was to find whether the presence or absence of the vertical cords and the central cord affected the sensitivity.

The results obtained by the 3 subjects are shown in table 3. Because a learning trend (training effect) was found for both subjects K. N. O. and V. J. E., only the results for N. W. are entirely reliable. The table shows the sequence in which the data were obtained and, also, the repeated data.

An inspection of table 3 shows that there is a small increase in sensitivity when the vertical cords are included with the cross in the field of view, but that the separation of these cords has little influence on it. Moreover, the addition of a central plumb line through the cross has little influence except when the other vertical cords are excluded; then a definite decrease in sensitivity is found. The latter was not anticipated, for it seemed as though the vertical cord through the cross would act as an axis for the apparent rotation of the cross.

An important result is that the ratio of the sensitivity to image size differences at axis 90 to that at axis 180 is of the order of 4 to 3. This indicates that the same precision of measurement on the space eikonometer cannot be expected for the measurements of image sizes in these two meridians.

C. Sensitivity to Changes in Declination.—A magnification of the image of one eye in an oblique meridian introduces, in addition to horizontal and vertical image size differences, a declination of the images of all vertical objects in space. This declination in the space eikonometer causes an apparent inclination of the cross about a horizontal axis through its center.

The geared 2 per cent meridional size lenses, described earlier in the paper, can be used to introduce a change in declination to offset any error present without altering the horizontal or vertical image size differences. These geared lenses always introduce an image size difference in the 45 and 135 degree meridians and have, therefore, no effect on the cross itself. The declination should affect the apparent inclination of the vertical images in the field of view. Because of the cyclofusional movements that usually take place, however, the cross actually appears inclined, the inclination being equal to that expected from the vertical cords. Thus, the sensitivity of the eyes to changes in declination can be measured by the apparent inclination of the cross. It was important to obtain data to show

TABLE 3.—*Tabulation of Data Which Indicate the Sensitivities Found for Changes in the Relative Sizes of Images in the Two Eyes in the Vertical Meridian (Axis 180) as Perceived in an Apparent Rotation of the Cross* *

Test Elements	Order in Which Data Were Taken	Standard Deviations		
		Subject N. W.	Subject V. J. E.	Subject K. N. O.
Cross only	2	0.110%	0.126%	0.099%
Cross and central white line	6	0.117%	0.156%	0.148%
Cross, front and rear cords separation 50 cm	5	0.091%	0.119%	0.124%
	8	0.099%	0.088%	0.085%
Cross, front and rear cords and single, center white cord, separation 10 cm	3	0.086%	0.140%	0.128%
Cross, front cords and single center white cord, separation 50 cm	1	0.081%	0.121%	0.139%
	7	0.085%	0.131%	0.097%
Cross, front and rear cords and single center white cord, separation 64.2 cm	4	0.077%	0.089%	0.102%

* Comparative data for different configurations used with the cross are shown. The visual distance was 3 meters.

whether the number of vertical cords in the field of view, especially the central line through the center of the cross, or the presence of triple cords through the center, as indicated in figure 5, affect the sensitivity.

The results for the 3 subjects are given in table 4. These data show that for 2 subjects there was a decidedly increased sensitivity to changes in declination when a vertical cord was

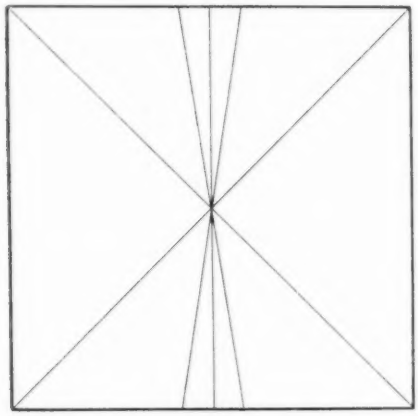


Fig. 5.—Illustration of the triple cord arrangement sometimes used in the space eikonometer. These cords lie in the plane of the cross and cross at its center.

placed through the center of the cross. With the triple cord arrangement, there was a tendency to even higher sensitivities. For the third subject, K. N. O., it did not make any difference. The explanation for this seems to be that the last subject was careful to use all possible spatial clues and compensated for the lack of the central

TABLE 4.—*Tabulation of Data Which Indicate the Sensitivities Found for Changes in Declination Between the Two Eyes as Perceived in an Apparent Inclination of the Cross* *

Test Elements	Order in Which Data Were Taken	Standard Deviations		
		Subject N. W.	Subject V. J. E.	Subject K. N. O.
Red cross, front and rear cords, triple center white cords.....	1	0.106°	0.076°	0.060°
Red cross, front and rear cords, single center white cord.....	2	0.104°	0.109°	0.081°
	5	0.070°	0.084°	0.077°
Red cross, front and rear cords, no center cord.....	3	0.194°	0.170°	.....
	6	.....	0.187°	0.075°
Red cross, single center white cord.....	4	0.092°	0.090°	0.074°

* Comparative data for different configurations used with the cross are shown. The visual distance was 3 meters.

cord, while the other 2 subjects, who had become accustomed to it, were not as careful to look for substitutes before making the judgments. A repetition of the third experiment of the sequence for subject V. J. E. demonstrates the repeatability of the data. While there is evidence of some training effects in the results for N. W. and V. J. E., it does not appreciably affect the interpretation of the data. The order of magnitude of these data is striking, for they indicate that in the majority of times in which the stimuli were presented the eyes were able to discriminate a declination angle of less than 0.1 arc degree (6 minutes of arc).

D. Modified Cross Configuration.—Other configurations of the cross are, of course, possible, and even practical for certain purposes.[4] Consider for example, the Maltese-like form shown in figure 6, consisting of four stretched cords all passing through the center of the configuration, each pair making an angle of 5 degrees with the diagonals of the square frame.

When a change in declination is introduced, the two "vanes" formed by the same two cords appear to turn in directions opposite each other about the 45 (or 135) degree axis (not unlike the blades of a propeller).

A study of the sensitivities of the 3 subjects to changes in declination as perceived on this cross was made as previously described. The results are shown in table 5. For comparison,

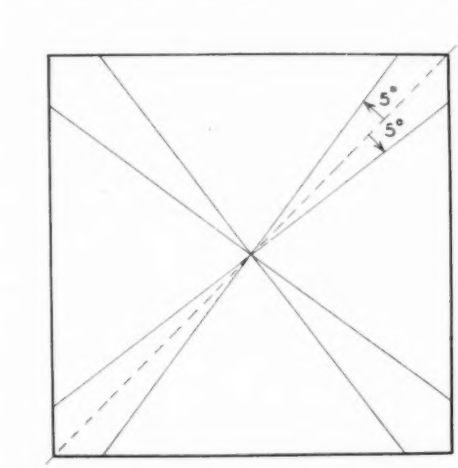


Fig. 6.—An alternate form of the cross which is highly sensitive to changes in declination.

the sensitivities found for the configuration of the regular cross are also included. The sequence of experiments is indicated in parentheses.

In the space eikonometer with the Maltese cross, the elements consist of the front and rear cords together with the cross, just as the regular space eikonometer is made up of simple cross, front and rear cords and single central cord. For this reason, the second and third horizontal columns should be used in comparison of the sensitivities with the configurations of the two

crosses. The results definitely show that a much higher sensitivity can be secured with the Maltese cross.

No explanation can be given for the increased sensitivities found for 2 of the subjects (V. J. E. and K. N. O.) when the front and rear paired cords were added as compared with those found with the Maltese cross alone.

TABLE 5.—*Tabulation of Data Which Indicate the Sensitivities to Changes in Declination as Recognized in the Apparent Distortion of the Maltese-Like Cross Configuration at a Fixation Distance of 3 Meters*

Test Elements	Standard Deviations N. W.	V. J. E.	K. N. O.
Maltese cross only.....	0.070° (1)*	0.098° (1) 0.094° (2)	0.079° (1) 0.083° (3)
Maltese cross, front and rear paired cords.....	0.079° (2)	0.066° (3)	0.060° (2)
Simple (two cord) cross, front and rear cords, single center white cord..	0.087°	0.105°	0.096°

* The numerals in parentheses indicate the order in which the data were taken.

E. Sensitivity Experiments on the Space Eikonometer for Near Vision.—Test elements of the space eikonometer, proportionally reduced in size, were also set up for measurements to be made at near vision (40 cm.). These elements were placed in a reading position normal to a visual plane with the eyes directed 20 degrees below the horizontal plane. Single fibers of triple strand silk thread were used to make the various configurations. The illumination at near vision was the same as that used at 3 meters. A large piece of black velvet placed 60 cm. behind the test elements served as background. All other parts of the instrument were the same as those previously described except that the axes of the test lenses were converged for the 40 cm. distance.

TABLE 6.—*Tabulation of Data Which Indicate the Binocular Sensitivities to the Space Eikonometer Test Elements for Near Vision and, for Comparison, the Equivalent Data Obtained for the 3 Meter Test* *

Subject	Sensitivities for	Regular Space Eikonometer, Visual Distance 40 Cm.		3 Meters
K. N. O.	Size differences, horizontal.....	0.087%		0.056%
	Size differences, vertical.....	0.096%		0.097%
	Declination change.....	0.205°		0.096°
N. W...	Size differences, horizontal.....	0.111%		0.074%
	Size differences, vertical.....	0.136%		0.085%
	Declination change.....	0.164°		0.087°
V. J.E.	Size differences, horizontal.....	0.089%		0.062%
	Size differences, vertical.....	0.213% 0.172%	Av. 0.192%	0.131% .....
	Declination change.....	0.239°		0.105°

* The values are the standard deviations, σ, obtained from an analysis of the method of constant stimuli.

The results of the same subjects in this test for near vision are shown in table 6, together with the results with the 3 meter instrument, for comparison.

In all cases but 1 (K. N. O. for axis 180), the sensitivities for near vision were found to be lower than those for distant vision. For the horizontal and vertical meridians, the ratio was of the order of 4 to 3. For sensitivities to changes in declination, however, the test for near vision was only half as sensitive as the 3 meter test. Again, the sensitivities for axis 90 (horizontal meridian) were definitely higher than were the sensitivities for axis 180 (vertical meridian).[14]

SUMMARY

This paper presents the results of a study of the stereoscopic thresholds of response for the space eikonometer. With this instrument, the aniseikonic error can be measured by the apparent orientation of the test elements as perceived by stereoscopic space perception. Three components of the aniseikonic error are measured: the image size differences in the horizontal meridian (axis 90) and in the vertical meridian (axis 180) and the error introduced in the normal declinations between the two eyes. The last will occur if the aniseikonic error includes a meridional image size difference at an oblique axis.

The stereoscopic sensitivities, as measured by the thresholds of discrimination, of 3 observers were determined by the method of constant stimuli under carefully controlled conditions and were expressed in terms of the standard deviation. For distant vision (3 meters) the results were as follows:

1. For the horizontal meridian (axis 90) the aniseikonic error is detected by an apparent difference in distance of two vertical plumb lines. For the clinical instrument, in which the lines subtend a 10 degree field (50 cm., separation at 2.4 meters), the standard deviation was of the order of 0.05 per cent in magnification of the images in the two eyes. This corresponds to an angular disparity between the images of the two eyes of 21 seconds of arc. Since the recognition of this disparity depends on the visual acuity of the retina at 10 degrees from the fovea, 21 seconds of arc is significantly high.

14. Polaroid vectographs of the test elements were also substituted for the actual threads and the sensitivities determined for both distant and near vision. In general, the sensitivities found were of the same order as those given above.

2. For the recognition of changes in the relative sizes of the images in the vertical meridian (axis 180), as detected in an apparent rotation of an oblique cross about a vertical axis, the standard deviation was of the order of 0.07 per cent in magnification of the images in the two eyes.

3. The sensitivity to changes in the vertical declination angle between the images in the two eyes, perceived in an apparent inclination of the oblique cross about a horizontal axis, as measured by the standard deviation was of the order of 0.1 degree.

Somewhat lower sensitivities (higher thresholds) were found for near vision, especially for the changes in the declination angles.

The results of this study of sensitivities emphasize the accuracy of the space eikonometer for the detection and measurement of aniseikonic errors.

Dartmouth Eye Institute.

CLINICAL DIFFERENTIATION OF EMBOLI IN THE RETINAL ARTERIES FROM ENDARTERITIS

ARTHUR J. BEDELL, M.D.
ALBANY, N. Y.

This paper is the summation of the study and correlation of many cases of sudden blindness caused by the abrupt closure of the central retinal artery, or a branch of the same. The conclusions have not been collected haphazardly but are presented after repeated examinations of the photographs and clinical histories. Textbooks, those proverbial exponents of repetitions, ancient and modern, rarely guide the student through the maze of generalizations to the correct diagnosis of changes in the retinal vessels. This is not said to disparage, for the compilation of any good textbook is an engaging, time-consuming task, but, rather, to ask the reader to start with an open mind for the reception of a new clinical differentiation between embolism of the central retinal artery and endarteritis. However, before the clearly defined groups are separated, it is essential to remember that there are cases of combined lesions, as, for instance, those of patients with severe hypertensive retinal changes who suddenly go blind as the result of a superimposed embolism. A source of constant confusion is spasm of the retinal arteries, which is, to my mind, a loosely applied term. Spasm plays an important role in some cases, and for that reason will be the subject of another paper, to be presented elsewhere.

The scope of this paper is strictly limited, and although pathologic proof is not presented, a sufficient number of reports are now available to warrant the conclusions here expressed. To emphasize further the visible differences in these cases, 2 cases of complete embolism are reported and photographically recorded from a few hours after the primary loss of vision to the terminal stage of complete optic nerve atrophy, with a narrow, threadlike retinal arterial tree. Particular attention is paid to the changes which at the onset take place in the circulation. These are greatest in the superior temporal vein, less frequent in the corresponding artery and still more rare in other branches. The alterations in the size, color and surface of the macular area, and the extent and intensity of the retinal edema are considered, and attention is drawn to the rapidity with which the disk becomes pale, loses the fine vessels on its surface and, finally, becomes white, with a shallow, complete atrophic cup. It must be repeated again and again that the retina is like the human body as a whole, and that vascular changes of similar intensity produce variable results. An unusually careful scrutiny of every case is necessary to observe the changes here described and few ophthalmologists record their observations in sufficient detail to be of value comparable to that of serial, color photographs of the fundus, and for that reason conclusions based on the analytic review of years of written reports are not always convincing. After the war, it will be possible to publish more colored reproductions, so that all who wish may examine them in the detail which they deserve.

ILLUSTRATIVE CASES

CASE 1.—A typical ophthalmoscopic picture of embolism, from eighteen hours after the primary obscuration of vision to the formation of complete optic nerve atrophy is well illustrated by the case of C. N., a 34 year old married man, who, twelve years before the beginning of his blindness, had acute rheumatic fever with endocarditis. Through the intervening years, he was seemingly free from any gross manifestations of heart disease and had never had any trouble with his eyes until Nov. 30, 1942, when suddenly the vision of the left eye began to fail and in one hour he was blind in that eye.

He was examined eighteen hours after the onset. Vision in the right eye was 20/30, and with correction of —0.50 D. sph. it was 20/20. The eye was normal externally, and there was evidence of pathologic changes in the fundus. The retinal vessels were completely normal.

In the left eye, vision was limited to faint perception of light. The pupil was 3.5 mm., regular and active. The media were clear, and the disk was sharply outlined. An oval zone, including the disk and extending a short distance beyond the macula, was both elevated and cloudy. The haze was greatest and the elevation most marked in the circummacular area, in the center of which was a small, bright red spot. The retinal veins were narrower than normal, and in places, particularly in the inferior temporal branch, the vessel was greatly contracted in portions of its course and slightly dilated in others, so that it looked like a streak hemorrhage, starting at the margin of the disk and extending toward 4:30 o'clock. The superior temporal veins showed distinct interruptions of the blood stream with spaces between the red cell groups, which

were pulsating, with a definite motion toward the disk. The arteries were definitely smaller than when the patient was first examined.

Twenty-four hours later, the edema was greater about the macular area, and the red spot was decreased in size and brightness. The haze surrounding the disk was increased in intensity but not in extent. More blood was passing through the superior temporal vein; but there were many interruptions in the stream, and the propulsion toward the disk was more easily discernible.

Two days later, the edematous perimacular swelling was flatter; the macular spot was brownish yellow, and the circulation through the superior temporal vein was almost completely reestablished.

One week later, there was narrowing of the lumen of the arteries, especially on the disk. There was no interruption in the current of the superior temporal vein and very slight reduction in the caliber of the inferior temporal vein. The disk was slightly paler than it had been, and the edema was decidedly decreased. From the macula there were radiating dark lines, breaking the clouding into segments. The red spot was larger than the previous week.

At the end of another week, the disk was distinctly pale. The caliber of the superior temporal vein was not uniform. The edema about the macula was less, and the red spot was larger.

Twenty-seven days later, the pallor of the disk was increased; the swelling of the retina was decreased, and the formerly raised, gray macular region was flat and granular white. The granules were best seen over the dark reddish center, where they appeared as yellowish, pinpoint specks. The edema about the disk had entirely disappeared, and the lumen of the superior temporal vein, as evidenced by the width of the blood column, was reduced.

At the end of ten days, the atrophy of the disk was manifest by its extreme pallor and the shallow excavation. The arteries were narrower than before, and the veins were constricted. There was no white or cloudy swelling about the macula, which was flat and dull gray. The fine, previously mentioned specks persisted.

Three months later, the appearance was typical of true embolism: a white disk, faint peripapillary pigmentation and, to the temporal side, partially absorbed choroid. The veins were about one-third their normal size and the arteries were thin threads devoid of plaques along their walls, but the macula was pink, with a very few pinpoint spots. The perimacular area was dark, slate gray. The eye was blind.

It is possible to theorize as to the cause of the embolus, for the patient had had endocarditis and from the valvular vegetations a plug may have broken loose. No other suggestive cause was discovered either in the history or on physical examination.

CASE 2.—In this case the patient was grossly devoid of cardiac disease. She had lost 30 pounds (13.6 Kg.) in the past two years, without any assignable cause. Suddenly, without any premonition, darkness spread over her left eye, and within eight hours the first photograph was taken.

The patient, a 65 year old woman, was first seen Feb. 6, 1942, when the vision in the right eye was 5/200, corrected to 20/20. The fundus was entirely normal.

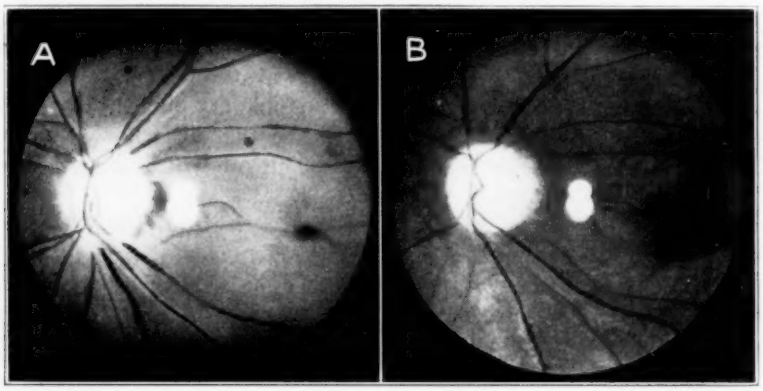


Fig. 1 (case 1).—*A*, embolism of the central retinal artery, with interrupted blood stream in the superior temporal vein and artery, oval edematous area and small macular red spot.

B, fundus seen in *A* five months after occlusion, showing white disk, narrow veins, attenuated arteries and granular macula.

Vision in the left eye was limited to faint light perception. The pupil was 3 mm. and reacted to light. The media were clear. The disk was clearly outlined, although partly covered by the retinal edema, which was very thick. A small uninvolved section of the retina, to the lower temporal edge of the disk, supplied by a cilioretinal artery, was normal pink. The edema was greatest about the macular region and extended slightly to the nasal side of the disk. The macula was dark red. There was interruption of the blood stream in the superior temporal artery, the paralleling vein and the inferior temporal artery.

The next day, the retinal edema was more uniform in texture and more dense. The macular spot, although still red, was reduced to about one eighth of its previous diameter. The interruptions in the blood stream in both the superior temporal artery and the vein were very evident. The blood cells were in constant oscillation, with motion toward the disk. The breaks in the blood current in the inferior temporal artery were less distinct.

Ten days later, the disk had lost some of its normal pink color. The edema was notably decreased but still

greatest about the macular region, where the central spot was twice its former size. The interruption of the blood stream was marked in the superior temporal vein, although there were no clear spaces between the cell groups. The circulation was restored in the artery. The volume of blood in some of the branches extending from the superior temporal vein down to the macula was unequal. The arteries showed a decided decrease in caliber; they were partially covered by the edema, and the inferior temporal branch had gray walls.

On the sixteenth day, there were striking changes; the disk was pale, the edema was practically confined to the perimacular region, the red spot had not increased in size since the last visit, the interruptions in the blood stream were absent and the vessels above the fovea were clearly seen.

On the twenty-fourth day, the only region in which the retinal swelling remained was about the macula, where the retina was almost homogeneous gray and nearly flat. Several fine, pinpoint specks were found in the area, and, as with all such lesions, they were best seen on the pink background of the macula. The arteries were more shrunken, so that in places they could be traced only with difficulty. There was no interruption of the blood stream. The disk was white and devoid of any fine vessels.

Thirty-six days after onset, the perimacular region was dark, flat and covered with scattered, yellowish, pinpoint specks. The macular area was larger than it had been; the arteries were smaller, with faintly visible gray sheathing.

On the sixty-first day, the disk was white, with a circumpapillary arc of pigment. The retina was mottled and dark, with a few specks between the macula and the disk. The arteries and the veins were decidedly smaller. There was complete loss of vision.

The color of the fundus background, darkly mottled, the age of the patient and the general conformation gave a different terminal picture than that noted in case 1. The small, almost triangular, area of the retina proximal to the disk at 3:30 and 4:30 o'clock was never edematous and never lost its color. This explains the retention of the very small field corresponding to that region. It will be recalled that this peripapillary clear portion has been referred to by other writers, and for that reason this evidence of its existence is presented.

An embolus of a branch of the retinal artery can be recognized ophthalmoscopically and proved pathologically. Clinically there are two types of embolus: one, which remains practically unchanged in size, and the other, in which there is an elongation as the plug becomes larger or the vessel proximal to it becomes thicker.

CASE 3.—An example of the enlarging plug is that of a woman 68 years old who had been under my observation for many years, with normal fundi. Twenty hours previous to the examination, on Oct. 20, 1938, she noticed a blur before her right eye and in a few minutes found that she could see only the lower portion of the objects at which she looked. Her only pertinent illnesses had been recent tonsillitis and chronic pyelitis. The systolic blood pressure was 180.

Vision in the affected eye, the right, was 18/200. Externally the eye was normal. The disk was clearly outlined, with a small central excavation. The retinal vessels in the upper half of the fundus were normal. In the lower branch of the artery, inferior to a bifurcate division, there was a white, bright, occluding plug, which seemed very slightly wider than the artery and perceptibly longer than wide. Distal to the occlusion, the artery was reduced in size but not abruptly collapsed, and there was no interruption in the blood stream. The portion of the fundus supplied by this artery was white, with an irregular outline extending to the lower portion of the macular area as a curved white border. This edema extended only slightly beyond the nasal side of the disk. There was a defect in the superior nasal quadrant of the visual field.

Four days later, there was no change either in the field or in the central visual acuity. The retina was unchanged in color and the plug unaltered in size.

At the end of fifty days, the arterial plug was slightly longer than when first noted, and the retinal edema had disappeared.

On the ninety-second day, vision was 20/100, corrected to 20/20 with +1.75 D. sph. The sector defect

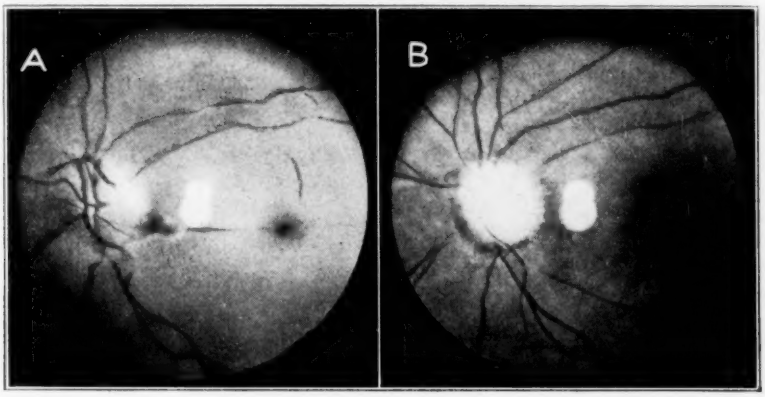


Fig. 2 (case 2).—*A*, embolism of the central retinal artery, with interrupted blood stream in both the superior temporal vein and the artery, broad oval of thick edema and small macular red spot.

B, fundus seen in *A* two months later, showing optic nerve atrophy, narrow arteries and veins and granular macula.

in the field persisted. The retina was of normal color. The vascular plug was about three times its original length, and the vessel distal to it remained unchanged in size and color.

In this case of sudden occlusion of the inferior retinal artery, the white plug was seen twenty hours after the onset; and at that time, when the localized retinal edema was pronounced, there was no interruption of the blood stream. Months later, when the plug had extended down the artery to more than twice its primary size, the vessel wall was unchanged, neither gray nor covered with white plaques.

In another case of embolus of the inferior branch, the white plug was grossly larger than the vessel, and the narrowing of the artery was very conspicuous.

CASE 4.—The patient, a 53 year old merchant, was not seen until a week after the sudden obscuration of the vision of the right eye.

A week later, the retina remained the same and the plug narrow.

Ten days later, the plug was practically unchanged, and the artery remained narrow.

At the end of eighteen days, the retina was of normal color.

Three months later, vision was 10/200. On the mottled retina the vessels were clearly seen; the inferior temporal artery was narrow on the disk and a short distance beyond the margin. The embolus was of the same size as when first seen. The lower half of the disk was pale, and the visual field had not enlarged.

In this case no systemic disease was discovered; no source of an embolus was uncovered, and there were no gross vascular changes. His systolic pressure was 140 aud his diastolic pressure in proportion.

This case was one of embolism, of unknown origin, permanently destroying the upper half of the visual field.

The first patient, with a branch occlusion, was seen early enough and followed at sufficiently short intervals for any breaks in the blood stream

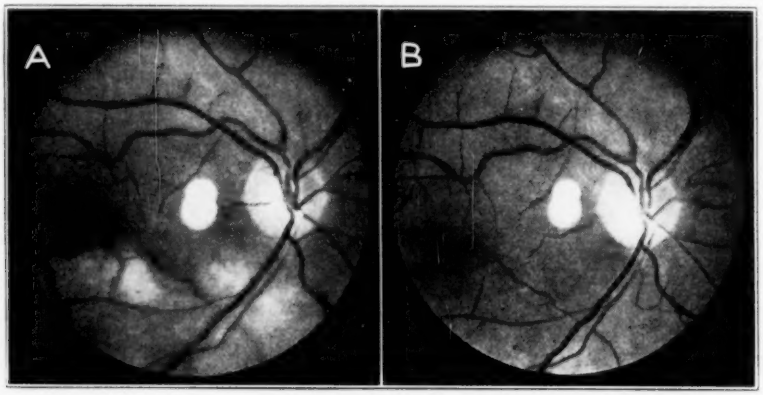


Fig. 3 (case 4).—*A*, embolism of the inferior temporal artery, with a visible plug in the artery on the disk. The vessel distal to the plug is narrowed. Cloudy retinal swelling of the inferior temporal quadrant is present.

B, fundus seen in *A* three months later, showing pale inferior temporal quadrant of the disk. The plug in the artery is unchanged, with narrowing of the inferior temporal artery. Complete resolution of the retinal edema has occurred.

Vision in the right eye was 5/200, corrected to 10/200 with + 1.50 D. sph. Externally the eye was normal. The disk was almost round and was clearly outlined. Just inferior to the bifurcation of the lower branch of the central retinal artery, the temporal division was closed by a small, glistening white mass, wider than the diameter of the vessel and about twice as long. Immediately distal to the plug there was marked contraction of the artery, which was evident for a distance of ⅓ disk diameter, where it gradually widened, but not to the diameter of the superior branches. The vessels in the upper half of the fundus were normal in relative size and distribution. Some thick, white, round-bordered areas of retinal edema outlined the lower margin of the macular region. The inferior vein was unchanged, and there was no discontinuity in the blood flow. The edema was scarcely perceptible in most of the lower half of the retina. There was an almost complete loss of the upper half of the visual field.

On June 26, 1944 the white retinal areas were decidedly less evident.

to have been observed if they were present. The second patient did not come under observation for several days, too late for interruptions to be evident. However, up to the time of this report I have neither photographed nor seen a segmentation of the blood column in a case of branch block.

To appraise accurately the difference betweeen an occlusion in a presumably normal artery and one in a previously damaged vessel, it is necessary to recheck carefully the details and, when possible, the photographs. The following cases of endarteritis will demonstrate the value of such a study.

CASE 5.—The first case was that of a man 51 years of age who, about twenty-four hours before I saw him, noticed a cloud cover his right eye. A few seconds later, he covered his felt eye and found that his right eye was sightless.

On Sept. 2, 1937 the right eye was normal externally. The fundus was white with the exception of the disk and macular region. The retinal edema was of variable thickness, so that the effect was one of a dappled, milky background. The margins of the disk were ill defined because the swollen retina bulged over the edges. There was a small central excavation. The retinal veins were reduced in caliber; in some places they were partly covered by the edematous retina, and in others they were dark red, patent streaks. The arteries were small, especially about the disk; and, like the veins, they were often completely hidden in the retina. The vessels about the red macular area stood out as pink threads on a white background. There was no intermittent current in either the arteries or the veins. The walls of the artery were gray, particularly the inferior temporal branch near the margin of the disk.

Two weeks later, the pallor of the disk was greater; the retinal edema was less, and the walls of the nasal branches of the artery were more manifest. On the inferior temporal artery, almost directly below the disk, there was a white plaque. Distal to this lesion the vessel was a bloodless gray thread for a distance of about ½ disk diameter, at which point the blood stream regained its full volume.

Two months later, vision was limited to perception of fingers at 1 foot (30 cm.) in a very small temporal field. The pupil was 4 mm., regular and active. The white disk was clearly outlined, with an arc of partial depigmentation at the temporal margin. The arterial tree showed prominent sclerotic patches; the longest one was in the superior temporal branch, but there were others along the inferior division. Several of the other branches were white walled and all of them decreased

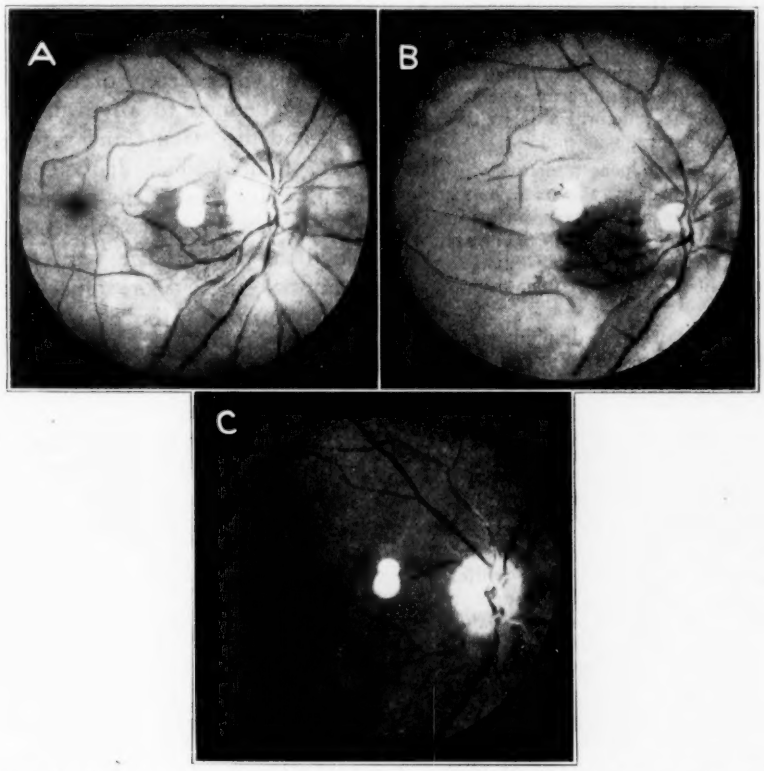


Fig. 4 (case 6).—*A* (March 14, 1942), endarteritis of the central retinal artery, with retinal edema twenty hours after onset. The clear portion is supplied by the cilioretinal artery. Changes in caliber appear in the arteries. The macular red spot is of normal size.

B, fundus forty-eight hours later. Retinal edema is much more extensive and intense; the macular red spot is a mere dot, and arterial changes are especially pronounced in the inferior temporal branch.

C, fundus twelve weeks after onset, showing complete optic nerve atrophy, very irregular caliber of the narrow arteries and white plaque in the wall of a sclerotic vessel. This is terminal stage of endarteritis.

The patient had not been under medical supervision, although his blood pressure was 210 systolic and 140 diastolic.

On the following day, there was a decrease in the size of the macular red spot.

Twenty-two days later, the edema of the retina had subsided but was still evident near the temporal side of the disk. The gross changes in the arteries were narrowing of the lumen and increased visibility of the wall with beginning plaques, best seen near the inferior margin of the disk. The macular area was red, granular and flat and the perimacular circulation well defined.

in size. The veins were smaller than normal. The macula was dark, with fine granular spots, and there was complete atrophy of the optic nerve, with a white nerve head. The patient died March 19, 1940.

The condition in this case was considered endarteritis for several reasons: first, the widespread primary edema, which involved the entire visible retina; second, the demonstrable old vascular changes, which were found immediately after the blindness and therefore were present

before the complete occlusion, and, finally, and most conclusively, the gross, white arteriosclerotic plaques, which were very different from the pale, yellow emboli, not only in their extent but in their position on the artery wall rather than occluding the lumen of the vessel.

The final case is instructive, for although it terminated in complete optic nerve atrophy, with white-walled arteries, there were peculiarities in the blood supply, the intensity of the retinal edema and the manner of its resolution.

CASE 6.—W. E. T., a 56 year old man, suddenly lost the sight of the right eye about twenty hours before the first examination.

On March 14, 1942, the vision of the right eye was limited to questionable perception of fingers. The pupil was 3.5 mm. and reacted promptly to light and in accommodation. The disk was clearly outlined and was of normal pink color, with a moderate-sized central excavation. An area to the temporal side of the disk, supplied by a cilioretinal artery, was pink.

The retinal edema was in places a dense white and in others appeared as a uniform gray haze. The macular red spot was of average size and clearly outlined. The veins were of normal size and distribution. The arteries, in many places covered by the gray retina, showed marked variations in caliber without segmentation of the blood column.

The patient was a stout, red-faced, plethoric person who had had high blood pressure for an unknown number of years. During this time he had two cerebral vascular spasms, when for a few days he was partially paralyzed. The blood pressure was 230 systolic and 120 diastolic. There was a slight trace of albumin. The roentgenogram disclosed a wide, tortuous aorta. The Wassermann reaction of the blood was normal.

The changes which took place in forty-eight hours were striking. The white clouding of the retina was more pronounced, and in places there were whiter, elevated mounds, of great density. The macula was a mere pink dot. Along the side of some of the finer arteries there were delicate streak hemorrhages. All of the retinal vessels were smaller than normal.

Three days later, the thick, white retinal cloud was intense. The disk was still pink. The arteries showed an increased visibility of the walls, most evident inferiorly. The macula was only a pinpoint, and the vessels surrounding the upper portion of it had an irregularity of lumen which could easily be mistaken for an interruption of the blood stream.

At the end of another week, the edema was disappearing. It was greatest about the macula, with broad side wings above and below it. The macular red spot had doubled in size but was smaller than when first examined. The disk was beginning to lose its color.

A week later, the retinal edema was less, remaining greatest about the macula and in a crescentic zone outlining the distribution of the superior temporal artery. The macula was red. There was an increased visibility of the artery, with considerable decrease in the lumen of several branches, especially the inferior temporal ones.

After another seven days, the disk was definitely white; the edema was decreased in intensity, and the macula remained a red spot.

Twelve weeks from the onset, there was an atrophic depression of the white disk, with irregular surrounding pigmentation. Arteriosclerotic white plaques were visible along the arteries, greatest over and proximate to the disk, as usual. Both the arteries and the veins were very narrow, the former mere threads. The macula was slightly redder than the surrounding fundus, and there were a few white granular specks on and about it.

This case illustrates the arteriosclerotic plaques, the density of the retinal edema and the manner of its disappearance in a patient with hypertension who died shortly afterward, from a cerebral vascular accident.

SUMMARY

In cases of embolism of the central retinal artery, an early sign is the interruption of the blood stream, usually in a vein, although it may be in both a vein and an artery. The site of predilection seems to be the superior temporal divisions. The zone of retinal edema is rather sharply defined to an oval, which includes the disk and the macula and rarely, if ever, extends more than 1 disk diameter beyond either or both. The size of the macular so-called cherry red spot, a misnomer, depends on the choroidal blood supply, on the type of fundus, whether albinotic, mottled or some other form, and on the intensity of the edema. At the onset the macular spot is unchanged in size; but as the retinal edema increases, it becomes smaller and smaller and may entirely disappear. Later, as the edema subsides, the macula becomes larger and larger. With the disappearance of the edema the color of the fundus is more or less uniform, and many fine, yellow-white, pinpoint dots are scattered over it. These are best seen where the fundus is reddest—the macular region.

Coincident with the decrease in the edema there is a progressive narrowing of both the veins and the arteries. The arteries finally become mere white threads, devoid of plaques on their walls. It is probable that in the case of a true embolism there are no retinal hemorrhages. At a variable time after the plugging of the artery, the optic nerve begins to lose its normal pink color, and eventually it becomes a sharply demarcated, atrophic, white disk.

In cases of endarteritis there is at the onset of the blindness always some evidence of preexisting arterial disease—an increased reflex from the vessels, increased visibility of the wall, irregularity of caliber, indentation of veins, minute aneurysms, exudates, hemorrhages or other signs of arteriosclerosis and hypertension. The interruption of the blood stream is much less constant than in embolism, and often what seems to be a fragmentation is a beaded vessel or one covered by the edematous retina. The retinal edema

is thicker than in the case of embolism and is often dappled, with patches of increased whiteness in the involved area. The edema is usually throughout the entire visible retina, and is not limited to a posterior oval region.

An embolism may be superimposed on endarteritis; and after the primary retinal edema has subsided, a minute white plug may be found in a distal branch of the artery.

CONCLUSIONS

In cases of embolism of the central retinal artery, the arteries become mere threads and the veins greatly reduced in size. The wall of the artery may be visible. The optic nerve is white, completely atrophic and sharply outlined.

In cases of endarteritis, the arteries are rarely as small as those found with embolism. There are always white plaques along their walls. The optic nerve atrophy is similar to that of embolism.

The retinal edema from embolism is limited to an oval area, which includes the disk and the macula. The edema in endarteritis usually involves the entire visible fundus.

These conclusions, based on the intensive study of photographs and fundi, are offered for serious consideration and are advanced in the hope that other observers may confirm them, so that eventually embolism can be more readily differentiated from endarteritis.

344 State Street.

AN UNUSUAL CASE OF HODGKIN'S DISEASE

SECOND REPORT *

JOHN WAITE AVERY, M.D.
AND
J. W. WARREN, M.D.
HOLLYWOOD, CALIF.

In a previous issue of the ARCHIVES, we made a preliminary report on the case of Mrs. M. W., then aged 50, in whom the disease involved the lymphatics of the bulbar conjunctiva of both globes, as well as various somatic glandular structures.

A lump in the cheek was first noticed by the patient in 1935 and was soon followed by flat, reddish growths on the eyeballs.

They were not tender and did not respond to medical treatment. About one year after their appearance they were excised, but in six months they again appeared on both globes in the same locations. Invasion of other glands was increasing, the patient was weak and losing weight; vision was poor and painful, and she had pronounced nasal occlusion.

The patient came under the care of one of us (J. W. A.) in April 1940 and was referred to the Malignancy Committee of the Hollywood Presbyterian Hospital. A biopsy of tissue from the inguinal glands was made. The committee and the laboratory concurred in the diagnosis of Hodgkin's disease, after Wassermann (Kolmer) and Kahn tests and an agglutination test for brucellosis gave negative results.

In May and June 1940, Dr. J. W. Warren gave a series of fifteen roentgen treatments to the globes and the affected glands, and a second series, of ten treatments, in November and December of the same year.

"Ten days after the first roentgen treatment the ocular lymphoid growths were thinner and somewhat bleached. In another eight days the eyes were markedly better, and all enlarged glands smaller. Nasal obstruction was no longer felt subjectively, and the pharyngoscope showed an open epipharynx. The improvement was general and constant."

Frequent examinations of the eyes over a period of a year showed no unfavorable changes. Normal vision was maintained with a moderate correction. "By March 21, 1941, the only glandular enlargements palpable were the one on the left cheek, which was small and was felt only on deep palpation; a cordlike condition of the upper anterior cervical chains, small but palpable . . . , and a few small, irregular stained areas on the left globe at the site of the extensive lymphatic infiltration."

Since the date of this report, November 1941, we have kept regularly in touch with the patient. In 1942 no roentgenographic or other treatments were given.

In May and June 1943, ten roentgen treatments were given to the somatic glandular areas only, not to the eyes. There was no evidence of a return of the former pathologic process, but the patient was somewhat asthenic, and the gastrointestinal tract was not normal. The latter conditions were finally determined to be caused by overwork, under unfavorable shop conditions.

A change to better surroundings brought a rapid improvement in her general health, including a gain of 15 pounds (6.8 Kg.). The patient remains in excellent health and has had only a few slight colds the past four years.

There has never been the slightest tendency to a recurrence of the lymphatic involvement of the bulbar conjunctivas. Both scleras are white; but the left, at the site of the former growth, appears slightly pebbly, and two small vessels traverse this area. The former leukoma, near the center of the left cornea, is now a faint nebula measuring about 3 by 4 mm. The nose and throat are in good condition. There still remains a small infiltrate in the left buccal muscle, freely movable, painless and found only on deep palpation.

The upper part of each anterior cervical chain of glands is slightly palpable only as a thin cord, probably the result of a sclerotic change, and the posterior chains cannot be felt.

The Malignancy Committee of the Hollywood Presbyterian Hospital reports as follows:

"Examination reveals no evidence of the former Hodgkin's disease. The eyes seem clear. There is still a small residual lump in the left cheek. Palpation of all the superficial lymph nodes in the groins, axillas and elbows gives negative results. There seems to be no palpable enlargement of the spleen."

The purpose of this report is not to offer a new therapeutic approach to Hodgkin's disease but simply to record an apparently unreported invasion of unusual lymphatic areas, to which roentgen radiation was applied, successfully thus far, and without injury to the delicate structures of the globes.

We hope to report again, perhaps after several more years.

7166 Sunset Boulevard.
1680 North Vine Street.

* Report as of Dec. 18, 1944.

1. Avery, J. W., and Warren, J. W.: An Unusual Case of Hodgkin's Disease: A Preliminary Report, Arch. Ophth. **27**:1019 (Dec.) 1941.

Clinical Notes

CARDINAL POINTS IN THE STATIC AND IN THE DYNAMIC EYE

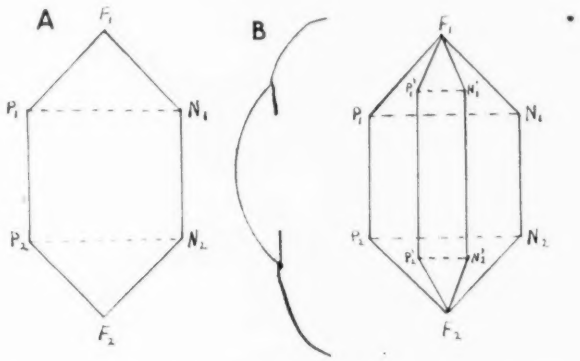
JOSEPH I. PASCAL, M.D., NEW YORK

The eye, like every compound optical system, has six important reference points (and planes) —the so-called cardinal points. These are the two principal focal points, designated as F_1 and F_2; the two principal points, P_1 and P_2, and the two nodal points, N_1 and N_2. Once these six points are located (in fact, even the first four suffice), calculations and constructions for image position, image magnification, etc., are easily made.

A scheme for remembering the relative distances between these cardinal points was previously published.[1] This method utilizes the well known benzene ring, on which the cardinal points are placed, as in *A* of the figure. Any two opposite sides which are parallel are equal. Thus if the distance between any two points is represented by the two corresponding letters, $P_1 F_1 = N_2 F_2$; $P_2 F_2 = N_1 F_1$; $P_1 P_2 = N_1 N_2$, and $P_1 N_1 = P_2 N_2$. The distance between the principal point and the corresponding nodal point, i.e., $P_1 N_1$ or $P_2 N_2$, is equal to the radius of the single refracting surface which can replace the whole ocular system. This potential radius is equal to the difference between the principal focal lengths.

Now, when the eye accommodates it becomes a different, specifically a stronger, optical system. This causes a displacement of all the cardinal points, so that they occupy different positions in the accommodated eye than they did in the non-accommodated eye. The shift in the cardinal points resulting from accommodation can be visualized and easily remembered by utilizing another benzene ring inside the original ring. This is a thinner and longer "ring," placed as in figure, *B*. I shall designate the principal and the nodal points of the accommodated eye as P_1'; P_2'; N_1'; N_2' (read P one prime, etc.).

It is known that since the accommodated eye is a stronger optical system, its focal distances must be shorter. A glance at the diagram (*B*) shows this, as F_1 is closer to P_1' than it is to P_1 and F_2 is closer to P_2' than it is to P_2. The distance between any two points may be represented by the two corresponding letters, and one sees that the distance between the principal points in the accommodated eye, P_1' and P_2', has increased, as has the distance between the two nodal points, N_1' and N_2'. The distance between the principal and the corresponding nodal point has decreased. That is, $P_1' P_2'$ is more than $P_1 P_2$; $N_1' N_2'$ is more than $N_1 N_2$; $P_1' N_1'$ is less than $P_1 N_1$ and $P_2' N_2'$ is less than $P_2 N_2$.



Scheme to show cardinal points (*A*) in the static (nonaccommodated) eye and (*B*) in the accommodated eye.

The equality of opposite parallel sides, of course, holds as good in the inner "ring" as in the outer "ring." Thus, $P_1' F_1 = N_2' F_2$; $P_2' F_1 = N_1' F_1$, and so on.

As a result of the increase of power, one knows that F_1 and F_2 must have moved closer to the cornea. The diagram shows that as a result of the accommodation the two principal points have moved farther apart, as have the two nodal points. Also, the principal and the corresponding nodal point have moved closer together.

When one visualizes the cornea to the left of the "ring" in *B*, one sees what happens during

1. Pascal, J. I.: A Memory Scheme for the Cardinal Points, Arch. Ophth. **22**:448 (Sept.) 1939.

accommodation. The principal points have moved farther away from the cornea, P_1 to P_1', and P_2 to P_2'; the nodal points have moved closer to the cornea, N_1 to N_1' and N_2 to N_2'. This movement of the principal and the nodal points brings the corresponding pairs closer together and shows the increased power of the system. For P_1 N_1 in the nonaccommodated eye and P_1' N_1' in the accommodated eye, each represents the equivalent radius of curvature of a single refracting surface which could replace the whole optical system.

The forward movement of the nodal points explains why the retinal image, irrespective of its clearness, is larger in the accommodated eye than it is in the nonaccommodated eye. The greater separation of the nodal points in the accommodated eye also shows that the displacement of the axial ray is greater in the accommodated eye than it is in the nonaccommodated eye.

Thus, the long benzene "ring" inside the regular "ring" shows at a glance the changes taking place in the position of the cardinal points when the eye accommodates and their significance. With the aid of the double "ring" these relationships can be easily remembered.

37 West Ninety-Seventh Street.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

RETINAL VASCULAR MICROMETRY AND ESSENTIAL HYPERTENSION

FERDINAND L. P. KOCH, M.D.
NEW YORK

Variations in the diameters of retinal vessels observable ophthalmoscopically in the eyes of normal and of hypertensive persons have drawn the attention of numerous workers since the first report of retinitis in nephritis was published by von Graefe,[1] in 1855; however, Liebreich,[2] in 1859, in his detailed description of "albuminuric retinitis," was the first to mention the existence of narrowed arterial vessels.

Frequent attempts have been made since the initial published method by Ruete,[3] in 1852, to obtain absolute measurements of the delineable ophthalmoscopically visible retinal structures in the living human eye (table 1). The basic goal has been the attainment of unquestionable accuracy by practical means. Numerous ingenious instruments and methods have been devised, all of which, however, in some manner combine a grid, graticule or micrometer scale with an ophthalmoscopic apparatus. The application of retinal photography, as advocated by Tengroth[4] and by Nordenson,[5] provides for the later, and more leisurely, analysis of structures that previously had undergone examination. Haessler and Squier[6] employed this method in 1931 in their study of the alterations in caliber of the retinal arterioles in persons with early hypertension. Puntenney,[7] more recently, has employed experimentally the method suggested by Lambert,[8] in 1934, by means of which direct microscopic examination of the living eye is facilitated by the use of a planoconcave contact glass. A miniature camera and a micrometer ocular attachment were used to provide photographic records of intraocular vessels under varying pharmacologic changes of environment.

The earliest investigators devised crude grids or graticules for use with reflecting mirrors and indirect ophthalmoscopy, but it was not until 1864 that the first measurements of the caliber of the arterial vessels of the human retina were published by Zander[9] (table 2). Subsequent technical improvements in the construction of ophthalmoscopes and their accessories made possible the eventual incorporation of the Morgan[10] gaticulle in the Keeler hand instrument for direct ophthalmoscopy. This graticule, or some modification of it, is the most readily utilizable hand grid measuring method now currently in use; its convenience obviates the necessity of transporting the patient to a more complicated, stationary apparatus.

In my own studies a modified Morgan graticule incorporated in a Keeler ophthalmoscope was used.[11] This instrument previously had been employed by Wagener[12] (1931) and by Cusick and Herrel[13] (1939) in their investigations.

From the Departments of Ophthalmology of Bellevue Hospital and New York University, and the Manhattan Eye, Ear and Throat Hospital.

1. von Graefe, A.: Ueber eine Krebsablägerung im Innern des Auges, deren ursprünglicher Sitz zwischen Sclera und Chorioidea war, Arch. f. Ophth. **2** (pt. 1): 214-224, 1855.

2. Liebreich, R.: Ophthalmoskopischer Befund bei Morbus Brightii, Arch. f. Ophth. **5** (pt. 2):265-286, 1859.

3. Ruete, C. G. T.: Der Augenspiegel und das Optometer für practische Aertze, Göttingen, Dieterich, 1852.

4. Tengroth, S.: Demonstration av mätningar av ögonbottnen, Hygiea **92**:828-829, 1930.

5. Nordenson, J. W.: Ueber Messungen am Augenhintergrunde, Ztschr. f. ophth. Optik. **19**:1-3, 1931.

6. Haessler, F. H., and Squier, T. L.: Measurements of Retinal Vessels in Early Hypertension, Tr. Am. Ophth. Soc. **29**:254-262, 1931.

7. Puntenney, I.: Effect of Certain Chemical Stimuli on Caliber of Retinal Blood Vessels, Arch. Ophth. **21**: 581-597 (April) 1939.

8. Lambert, R. K.: (*a*) A Method for Study of the Retinal Circulation, Arch. Ophth. **12**:868-873 (Dec.) 1934; (*b*) Studies of the Retinal Circulation by Direct Microscopy, Am. J. Ophth. **18**:1003-1013 (Nov.) 1935.

9. Zander, A.: The Ophthalmoscope: Its Varieties and Uses, translated by R. B. Carter, London, Robert Hardwicke, 1864, p. 101.

10. Morgan, O. G.: A Retinal Graticule, Brit. J. Ophth. **11**:339-341 (July) 1927.

11. Koch, F. L. P.: Retina in Systemic Hypertension: A Clinical Study of the Caliber of the Retinal Arterioles and the Retinal Arterial Diastolic Blood Pressure, Arch. Ophth. **26**:565-581 (Oct.) 1941.

12. Wagener, H. P.: Ocular Changes Following Cervicothoracic Sympathetic Ganglionectomy, S. Clin. North America **11**:867-873, 1931.

13. Cusick, P. L., and Herrel, W. E.: Retinal Arteriolar Changes as Part of an Induced General Vasospastic Reaction: Effect of Tobacco and Cold, Arch. Ophth. **21**:111-117 (Jan.) 1939.

TABLE 1.—*Micrometry of Intraocular Structures: Chronologic Development*

Year	Author	Measuring Device Employed	Unit Taken as Constant
1852	Ruete [3]	Graticule; indirect ophthalmoscopy	
1854-55	Ruete, C. G. T.: Lehrbuch der Ophthalmologie, ed. 2, Braunschweig, Friedrich Vieweg u. Sohn, 1855, vol. 1	Graticule; indirect ophthalmoscopy	
1854	Donders, F. C.: Arch. f. Ophth. **1** (pt. 2): 75-105, 1854-1855	Approximating pointers; micrometer scale; indirect ophthalmoscopy	
1857	Schneller, J. J. M.: Arch. f. Ophth. **3** (pt. 2): 120-186, 1857	Approximating pointers; linear measurements; indirect ophthalmoscopy	
1878	Landolt, E.: Mikrometrie des Augengrundes, in Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1904, vol. 4, pt. 1, pp. 48-51	Graticule; indirect ophthalmoscopy; formulas	Disk = 1.5 mm.
1907	Fortin, E. P.: Arch. d'opht. **27**: 397-405 (June) 1907	Entoptoscopy	
1922	Lo Cascio [16]	Graticule; heliometer	
1924	Henriksson [17]	Split prism ocular	
1926	Bretagne, M. P.: Ann. d'ocul. **163**: 192-198, 1926	Graticule; formulas	
1926	Dufour, M.: Ann. d'ocul. **163**: 198-201, 1926	Graticule; formulas	
1930	Tengroth [4]	Retinal photography, micrometer scale, microcomparator	Structures measured in 0.01 disk diameter
1930	Margotta, G.: Ann. di ottal. e clin. ocul. **58**: 676-700 (Aug.-Sept.) 1930	Graticule, formulas	Disk value fixed
1931	Nordenson [5]	Retinal photography, stereomicrometer scale	Structures measured in 0.01 disk diameter
1931	Haessler and Squier [6]	Retinal photography; Gaertner comparimeter microscope	
1932	Lambert, R. K.: Arch. Ophth. **7**: 440-443 (March) 1932	Ophthalmoscopic grid	
1934	Spinelli, F.: Klin. Monatsbl. f. Augenh. **92**: 93-107 (Jan.) 1934	Modified perimeter; micrometer or graticule scale	Disk value fixed
1934	Lambert [8a]	Retinal photography; contact glass; micrometer scale	
1935	Lambert [8b]	Retinal photography; contact glass; micrometer scale	
1939	Puntenney [7]	Lambert method	
1940	Cotton, Lewis and Egenhofer [23]	Lobeck method, corrected formulas	Disk = 1.43 mm.
1940	Cotton and Rosin [24]	Lobeck method, corrected formulas	

TABLE 2.—*Caliber of Normal Retinal Arterioles*

Year	Author	Measuring Device Employed	Unit Taken as a Constant	Point at Which Measurements Were Made	Caliber of Arterioles, Mm.		
					Nasal	Temporal	All Vessels
1864	Zander [9]	Graticule					0.0564 to 0.1128
1907	Dimmer, F.: Klin. Monatsbl. f. Augenh. **45**: 296-329, 1907	Graticule; photography	Known diameter of papilla	At disk margin			0.093
1919	Hess, C.: Arch. f. Augenh. **84**: 1-10, 1919	Graticule	Disk = 1.5 mm.	At disk margin			0.100
1926	Lo Cascio [18]	Graticule; heliometer principle	Dick = 1.5 mm.	At disk margin			0.110 to 0.120
1931	Wagener [12]	Morgan graticule; Keeler ophtnalmoscope		½ disk diameter from disk margin	0.050 to 0.060	0.100 to 0.060	
1934	Lobeck [19]	Gullstrand ophthalmoscope; Lobeck heliometer	Dick = 1.5 mm.	At disk margin			0.140 to 0.150
1936	Neame [14]	Morgan graticule; Keeler ophthalmoscope	Disk = 1.5 mm.	At disk margin			0.086 to 0.094
1937	Lobeck [20]	Gullstrand ophthalmoscope; Lobeck heliometer	Disk = 1.5 mm.	At disk margin			0.088 to 0.134
1937	Kühn [21]	Lobeck's method	Disk = 1.5, 1.6 and 1.7 mm.	At disk margin			0.086 to 0.138
1937	Badtke [22]	Lobeck's method	Dick = 1.5 mm.	At disk margin		0.90 to 0.112	
1939	Rucker [25a]	Prism displacement method	Assumes nodal point of 17 mm.	½ disk diameter from disk margin			0.070 to 0.140
1939	Cusick and Herrel [13]	Morgan graticule; Keeler ophthalmoscope		½ disk diameter from disk margin	0.050 to 0.060	0.100 to 0.120	
1940	Cusick, Benson and Boothby [25b]	Morgan graticule; Keeler ophthalmoscope; Rucker prism displacement method		½ disk diameter from disk margin			0.072 to 0.102
1941	Koch [11]	Morgan graticule; Keeler ophthalmoscope		1 disk diameter from disk margin	0.047 to 0.124	0.062 to 0.140	0.047 to 0.140

The graticule is cross lined into millimeter squares, and equidistant cross lines subdivide these into squares of 0.2 mm. each. These individual squares, when focused on the retina in cases of emmetropia, bound an anatomic retinal surface area which has been calculated by the maker to measure 0.186 mm. on each of its four inner margins. On ophthalmoscopic examination, the size of these smaller squares increases in cases of hyperopia, when projected on to the retina, and decreases in cases of myopia; for example, any side of such a square is equal to an anatomic distance of 0.190 mm. at + 3 D. but only to 0.180 mm. at — 3 D.

The graticule is interposed in the beam emanating from the electric ophthalmoscope lamp and is focused sharply on the retina by adjustment of a separate attachment on the instrument. The spherical refractive power of the eye under examination is indicated as in other standard ophthalmoscopes. The maker has furnished a correction table for the various dioptric strengths necessarily employed in bringing into clear view the intraocular structures being measured. These strengths range from + 10 D. through 0 to — 10 D., inclusive. Since retinal vessels occupy only a portion of any small square of the graticule projected on the retina, fractional estimation of their relative size must be made. A monogramic table of equivalents was devised to facilitate the conversion of these values to microns (table 3).

TABLE 3.—*Table of Equivalents for Modified Morgan Graticule in Keeler Ophthalmoscope*

Dioptric Strengths	Value of Graticule Squares in Mm.	Fractional Estimation of Squares and Corrected Values in Microns					
		⅕	¼	⅓	½	⅔	¾
+10.00	0.192	38.40	48.00	64.00	96.00	128.00	144.00
+ 8.00	0.1916	38.32	47.90	63.87	95.80	127.73	143.70
+ 6.00	0.1913	38.26	47.83	63.77	95.65	127.53	143.48
+ 4.00	0.191	38.20	47.75	63.66	95.50	127.32	143.25
+ 3.00	0.190	38.00	47.50	63.33	95.00	126.66	142.50
+ 2.00	0.189	37.80	47.25	63.00	94.50	126.00	141.75
+ 1.00	0.188	37.60	47.00	62.66	94.00	125.32	141.00
0.00	0.186	37.10	46.50	62.00	93.00	124.00	139.50
— 1.00	0.184	36.80	46.00	61.33	92.00	122.66	138.00
— 2.00	0.182	36.40	45.50	60.66	91.00	121.32	136.50
— 3.00	0.180	36.00	45.00	60.00	90.00	120.00	135.00
— 4.00	0.178	35.60	44.50	59.33	80.00	118.66	133.50
— 6.00	0.172	34.40	43.00	57.33	86.00	114.66	129.00
— 8.00	0.164	32.80	41.00	54.66	82.00	109.32	123.00
—10.00	0.150	30.00	37.50	50.00	75.00	100.00	112.50

The instrument employed by Neame [14] (1936) was the Keeler ophthalmoscope in which he had incorporated his modification of the Morgan graticule. The graticule differs from that originally devised by Morgan in that squares and guiding lines of various sizes permit of a more versatile use. Another modification, introduced by the maker in the more recent "Decagon" model, has been studied by Berens and McLaughlin.[15] The value for each smaller square in this instrument in the emmetropic eye is equal to 0.1408 mm.; also, there is a larger central square in which lines of varying thickness have been etched, so that retinal structures as small as one third of a square may be measured. It is thought that errors of measurement dependent on estimation of size are decreased by the use of this innovation; however, there is as yet no confirmation of this statement.

Wagener,[12] in 1931, in measuring the retinal arterioles before and after cervicothoracic sympathetic ganglionectomy, and Cusick and Herrel, in 1939,[13] in their study of the changes in the retinal arterioles as part of an induced general vasospastic reaction, appear to have been the first to select a point peripheral to the margin of the optic disk at which to determine the caliber of the vessels under observation. They measured the diameter of the retinal arterioles at approximately ½ disk diameter distal to the margin of the papilla. That this is of some significance already has been pointed out with regard to whether the retinal arterial vessels are arteries or arterioles. It will be mentioned subsequently.

Lo Cascio,[16] in 1922, appears to have devised a formula based on principles employed in

14. Neame, H.: A Method of Estimating the Calibre of Retinal Arteries in the Living Eye by Means of the Ophthalmoscope, Illustrated by Results in Some Normal and Pathological Cases, Tr. Ophth. Soc. U. Kingdom **56**:155-162, 1936.

15. Berens, C., and McLaughlin, C. K.: The Keeler Decagon Ophthalmoscope as a Refractometer, Am. J. Ophth. **17**:402-416 (May) 1934.

16. Lo Cascio, G.: Un nuovo metodo per la determinzione della grandezza reale di tratti di fondo oculare a mezzo dell'oftalmoscopio, Riv. d'ottica **2**:51-64 (July) 1922.

astronomy, which, when applied to observation of the retinal structures with Gullstrand and Thorner ophthalmoscopes, anticipated the later work of Henriksson.[17] Lo Cascio,[18] in 1926, published a few measurements of the caliber of retinal arterial vessels at the margin of the disk in a small group of persons, of whom some were normal adults and some had chronic renal disease.

Lobeck,[19] in 1934, described his method and apparatus for the measurement of ophthalmoscopically visible retinal structures. The device is based on the principles of heliometry and is installed in the reflex-free, simplified Gullstrand ophthalmoscopé. Henriksson,[17] in 1924, had suggested their application, but, unlike the modification offered by Lobeck,[19] Henriksson's instrument was so constructed that the lens of the objective was split, while in Lobeck's the ocular was split. The earlier work of Lo Cascio[18] appears to have been unknown to these two authors.

It is possible with his apparatus, according to Lobeck,[20] writing in 1934 and in 1937, to measure the optic papilla with absolute accuracy and, by splitting the ocular and making positional adjustments of its axis, to determine the diameter of the retinal vessels at any point on the disk or at its margin by a direct reading from the scale mounted on the ocular which Nonius incorporated in the apparatus. It is necessary, however, to accept as a constant unit the figure of 1.5 mm. as representing the horizontal diameter of the disk. Lobeck cited several authors to support his contention that this is the mean anatomic size of the nerve head.

Kühn,[21] in 1937, employed the Lobeck heliometer and measured the papillas and the retinal vessels at the margins of the disks of 100 normal adults. He assumed arbitrary successive papilla diameters of 1.5, 1.6 and 1.7 mm. in order to compare the probable accuracy of the measurements he made of the retinal vessels. He calculated first the true values of the measured relative values, working from a basic absolute horizontal diameter of the papilla of 1.5 mm., as did Lobeck. His conclusions tended to confirm the results of the latter, and he came to the conclusion that in normal adults retinal arteries that measured less than 86 microns or more than 138 microns in caliber at the rim of the papilla and retinal veins that measured less than 100 microns or more than 159 microns at the same location were narrower and wider, respectively, than normal.

Badtke,[22] in 1937, also using the Lobeck apparatus, obtained essentially the same range of measurements of retinal arterial vessels in normal persons. Cotton, Lewis and Egenhofer,[23] however, in 1940, employed a modification of the method of calculation described by Lobeck in that they applied a factor for the refractive correction of each eye of their subjects to a formula from which they believed that the exact magnification of retinal structures observed with the Lobeck device could be determined. They expressed the opinion that the mean figure of 1.5 mm. for the horizontal diameter of the disk, as proposed by Lobeck and accepted by Kühn and by Badtke, permitted too large an error, since they found in a small group of cases that the true disk diameter varied from 1.2 to 1.7 mm. The mean value obtained was 1.43 mm.

These authors stated the belief, also, that it was necessary to measure the diameter of each retinal vessel rather than only one or a few of them because an additional factor of error would be introduced if this were not done. They gave no figures, however, for the diameters of the vessels measured but calculated in square millimeters the entire retinal vascular bed at the margin of the disk by measuring the caliber of all the vessels, both arterial and venous, in each eye at that location. These figures, they stated, were free from any assumed values.

They concluded that the total retinal vascular bed probably was diminished in certain deteriorative mental diseases. It should be pointed out, however, that Kühn found smaller vascular diameters when correcting his figures on the basis of a papilla diameter of 1.6 mm. than when he used larger disk sizes. In the absence of recorded actual values for vascular diameters, it is reasonable to suppose that had Cotton, Lewis and Egenhofer given these figures, they would have

17. Henriksson, V.: Oftalmomikrometriska försök, Hygiea **86**:887, 1924.

18. Lo Cascio, G.: Ricerche cliniche, anatomo-patologiche e patogenetische sulla neuroretinite nefritica, Ann. di ottal. e clin. ocul. **54**:3-64 (Jan.) 1926.

19. Lobeck, E.: Ueber Messungen am Augenhintergrunde, Arch. f. Ophth. **133**:152-165 (Nov.) 1934.

20. Lobeck, E.: (*a*) Ueber den Durchmesser der Netzhautgefässe am gesunden und kranken Menschen, Arch. f. Ophth. **136**:439-456 (Feb.) 1937; (*b*) footnote 19.

21. Kühn, W.: Ueber Messungen am Augenhintergrunde, Arch. f. Ophth. **138**:129-148 (Oct.) 1937.

22. Badtke, G.: Kalibermessungen an den Netzhautgefässen bei Hochdruck- und Nierenkranken, Klin. Monatsbl. f. Augenh. **99**:655-669 (Dec.) 1937.

23. Cotton, J. M.; Lewis, N. D. C., and Egenhofer, A. W.: Vascular Bed of the Retina in Mental Disease, Arch. Neurol. & Psychiat. **43**:891-900 (May) 1940.

been smaller generally than those of Kühn, since these three co-authors in their study determined the anatomic value of the disk to be 1.43 mm. Cotton and Rosin [24] further refined the formula method and concluded that measurements thus obtained probably were reliable to 0.01 mm.

There should be mentioned here the prism displacement method of Rucker [25] (1939), which assumes a nodal point of approximately 17 mm. and, therefore, a lesser magnification of the fundus than that of 14 to 15 diameters which is believed to be obtained with the standard hand ophthalmoscope.[26] His values for the caliber of the arterial vessels of the human retina compare favorably with those obtained by other workers with other methods.

It is difficult, however, to compare the figures obtained by the various investigators, since dissimilar instruments have been employed, methods and subjects have varied greatly and the sites at which the vessels were measured also have been unalike. Certain of the workers measured only some, or not all, of the vessels. All the figures obtained undoubtedly reflect to some degree the intangible factor inherent in the clinical fact that not only the observer but the subject was not a precision machine. Therefore, no matter how carefully and patiently the measurements were made, some deviations from absolute accuracy probably have occurred. Thus, it would appear that the recorded measurements of the diameters of retinal vessels in the living eye probably should be considered in the main as no more than approximately or reasonably accurate.

A comparison of the average or ranges of the figures for all arterial vessels, whether measured with the graticule instruments or with the heliometer devices, reveals no notable discrepancy if it is recalled that the use of the latter necessitates the measurement of the vessels at the margin of the papilla, where the diameters are readily seen to be greater than when observed more peripherally. Thus, all of the retinal arterial vessels in the vicinity of the nerve head in normal persons, with no systemic or local vascular disease, are found to range in caliber, as determined from all available published figures, from 50 to 120 microns when measured with the graticule and from 86 to 150 microns when measured with the heliometer (table 2).

24. Cotton, J. M., and Rosin, S.: Measurement of the Structures of the Fundus, Arch. Ophth. **23**:1146-1156 (June) 1940.

25. (*a*) Rucker, C. W.: Personal communication to the author, 1939. (*b*) Cusick, P. L.; Benson, O. O., Jr., and Boothby, W. M.: Effect of Anoxia and of High Concentrations of Oxygen on the Retinal Vessels: Preliminary Report, Proc. Staff Meet., Mayo Clin. **15**: 500-502 (Aug. 7) 1940.

26. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1933, vol. 1.

ANATOMY AND PHYSIOLOGY OF THE ARTERIAL VESSELS OF THE RETINA

The ophthalmic artery in man branches directly from the internal carotid artery and is considered by some authorities virtually a branch of the circle of Willis. The internal carotid artery narrows immediately after it gives off the ophthalmic artery. The latter averages 1.5 mm. in diameter while the former averages 5.4 mm. in diameter just proximal to the branching off of the ophthalmic artery and 3.8 mm. just distal to that point. A high pressure in the ophthalmic artery is believed to be maintained by this reduction in caliber of the internal carotid artery.[27]

The central artery of the retina arises from the ophthalmic artery on the entrance of the latter into the orbit. The former then traverses the lateral surface of the optic nerve until it enters the nerve, usually at a distance of 10 to 15 mm. posterior to the globe. The artery bifurcates within the intraocular portion of the optic nerve into a superior and an inferior papillary artery. Ophthalmoscopically, these two branches are not always fully visible because they exhibit remarkable variability in their anatomic lengths and in their points of emergence on to the papilla; however, they rarely extend beyond the rim of the optic disk. Each of these two arteries divide, in turn, to give rise normally to temporal and nasal arterial branches, which traverse the nerve fiber layer of the retina and supply the retinal quadrants to their peripheries.

Friedenwald,[28] in considering the role of arterial vessels of the retina, stated: "It is of no interest whether these vessels are arteries or arterioles." Wagener [29] pointed out, however:

> Perhaps of first importance in the interpretative study of the retinal arterial vessels is the decision as to whether they should be regarded as arteries or arterioles.

The central artery of the retina does not differ

27. Whitnall, S. E.: The Anatomy of the Human Orbit and Accessory Organs of Vision, ed. 2, London, Oxford University Press, 1932. Friedenwald, J. S.: Retinal Vascular Dynamics, Am. J. Ophth. **17**:387-395 (May) 1934.

28. Friedenwald, J. S.: The Pathology of the Ocular Changes in Nephritis and Hypertension, in Berglund, H., and Medes, G.: The Kidney in Health and Disease, Philadelphia, Lea & Febiger, 1935, pp. 638-664.

29. Wagener, H. P.: The Nature and Significance of the Retinal Lesions Associated with Hypertensive Disease, Tr. Am. Acad. Ophth. **44**:54-74, 1939.

essentially from other vessels of similar size in its course through the orbit and the optic nerve; however, after the artery passes through the lamina cribosa of the optic nerve, the arterial coat diminishes rather abruptly to about one third of its previous thickness. This transition normally is less obvious between the central artery and the papillary arteries than it is between the latter and the retinal arterial vessels proper. These smaller vessels diminish progressively in caliber to capillary size.

Friedenwald [28] has observed that the reduction in caliber is due largely to the diminution of the media, although the adventitia becomes thinner and looser in structure. The histologic demonstration of the presence of smooth muscle fibers in the normal retinal arterial branches is difficult, but the presence of contractile elements on both clinical and experimental grounds is conceded by this worker. The elastic lamella at first is preserved but becomes somewhat reduced in density until it no longer can be seen in the more distal secondary branches and in the tertiary branches. The intima remains unchanged. The veins and the arteriolar and venous capillaries normally are differentiated histologically from precapillary arterial vessels of any size by their lack of smooth muscle cells and the presence of an internal elastic lamella.

The retinal vessels sustain an internal pressure approximately equal to that sustained by vessels of a similar order of size elsewhere in the body, but the pressure in the terminal arterioles most probably is somewhat higher than the average elsewhere. The intraocular pressure of some 20 mm. of mercury, however, exerts an external supportive force on the intraocular vessels. Friedenwald [28] stated:

> It is perhaps as a response to this relief from internal strain that the unusual delicacy of the retinal arterial wall is to be understood. This delicacy of structure may explain the unusually great local effects of changes in blood pressure if these local effects are truly disproportionate to those in other organs and not merely more easily recognizable.

He also suggested that relative malnutrition of the walls of the retinal arterial vessels may result in fibrous thickening of the media and adventitia, since there exist no vasa vasorum in these small vascular channels.

The size of the arterial vessels in the retina, as well as those of similar caliber elsewhere in the body, has interested many observers. Kernohan, Anderson and Keith [30] and their co-workers classified small arterial vessels of diameters ranging from 25 to 125 microns as arterioles. Others have placed the upper limit of arteriolar diameters at 150 microns, while Bell [31] stated his opinion that this limit should not exceed 40 microns. The muscle and the internal elastic lamina, however, already have been reduced to a minimum in vessels of the latter caliber, and such vessels should be considered precapillary arterioles.

These workers studied fixed tissues stained with standard or special dyes and examined microscopically. Only vessels that were cut to true cross sections were measured. A monocular micrometer for incorporation in a standard microscope was used. The distinction between arteries and arterioles in their studies, therefore, is largely an arbitrary one and is based on the diameter of the vessel rather than on other criteria, since there exists no true anatomic distinction, such as that between the structure of venous and of arterial vessels of precapillary size or larger.

Ophthalmoscopically, measurements of the retinal arterial vessels, whether made with the graticule or with the heliometer, have been taken in the main at the margin of the optic disk. The previous exceptions have been those made by Wagener [12] and by Cusick and Herrel.[13] These workers have referred to the retinal arterial channels measured by them as arterioles; the other investigators have termed these vessels arteries, although their recorded measurements of these vessels at the margin of the papilla, or just immediately peripheral to that point, in the living human eye do not exceed the arbitrary upper limit set for arteriolar caliber. Inadvertently, the papillary arteries occasionally may have been measured.

It would appear, then, that the retinal branches of the central artery of the retina should be designated as arterioles, since they are within the limits of size of arterioles in general. Wagener [29] pointed out that this is of clinical importance when he stated, "Lesions observed in the retinal arterial vessels should be considered as indicative of systemic arteriolosclerosis, rather than of arteriosclerosis."

COMMENT

Wagener and Keith,[32] in their recent review of diffuse arteriolar disease with hypertension and the associated retinal lesions, stated: "Any elevation of systemic blood pressure is probably

30. Kernohan, J. W.; Anderson, E. W., and Keith, N. M.: The Arterioles in Cases of Hypertension, Arch. Int. Med. **44**:395-423 (Sept.) 1929.

31. Bell, E. T., cited by Wagener.[29]

32. Wagener, H. P., and Keith, N. M.: Diffuse Arteriolar Disease with Hypertension and the Associated Retinal Lesions, Medicine **18**:317-430 (Sept.) 1939.

accompanied by a rise of blood pressure in the retinal arterial system." Baillart [33] and his followers and Suganuma and others [34] have demonstrated that the rise in the retinal intra-arterial pressure under varying conditions may or may not be proportional to the elevation of pressure in the brachial arteries. The coincidental and associated changes, both physiologic and structural, that occur in the retina and in its vessels with elevation of the systemic and, presumably then, of the retinal blood pressure lend themselves to early recognition by the usual ophthalmoscopic methods, and information of considerable clinical value may be obtained by careful examination. This interpretative procedure is valuable in differentiating the various types of systemic vascular disease in which elevations of blood pressure occur. Wagener and Keith [32] stated:

> If it can be assumed, as seems logical, that the visible reactions in the retinal vessels are similar, though at times disproportionate, to the invisible reactions taking place in vessels of similar size throughout the body, it is obvious that an explanation of the mechanism of these reactions in the retina will go far toward solving the mechanism of diffuse vascular disease. This phase of ophthalmoscopy is of particular interest to the internist.

It is now generally accepted that widespread increased peripheral resistance to blood flow in hypertensive, vascular renal disease results in an elevation of systemic blood pressure and that the arterioles, in the main, are the seat of this increased resistance. Richard Bright,[35] in 1836, in speculating on the cause of cardiac hypertrophy associated with renal disease, suggested that the two most logical explanations for the apparently increased work that the heart had performed were "either that the altered quality of the blood affords unwonted stimulus to that organ immediately; or that it so affects the minute and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system."

One of the first to suggest that there was present a vascular factor in addition to the renal lesion in Bright's disease was Johnson,[36] in 1868. He stated:

> In every fatal case of chronic Bright's disease with hypertrophy of the left ventricle there has been decided hypertrophy of the arterial walls in most of the tissues examined, not only in the kidneys, but also in the skin, the intestines, the muscles, and the pia mater. . . . The facts hitherto observed all point to the conclusion that hypertrophy of the walls of the small arteries is a result of their continued overaction in opposition to the heart.

Johnson, however, as did Bright, believed that the primary disease is in the kidney.

Gull and Sutton, [37] in 1872, stated the opinion that the pathologic lesions observed at autopsy in the heart and in the kidney in cases of chronic Bright's disease are secondary to primary disease of the peripheral capillaries and arterioles. There still is no unanimity, according to Keith,[38] concerning which of these two points of view approaches the truth.

Mahomed,[39] in 1879, expressed the opinion that high arterial pressure could exist in adults who were less than 30 years of age and did not exhibit any other physical signs of disease and/or structural changes in the vessels. He termed this condition the "pre-albuminuric stage" of Bright's

33. Baillart, P.: Circulation artérielle rétinienne: Essais de détermination de la tension artérielle dans les branches de l'artère centrale de la rétine, Ann. d'ocul. **154**:257-271, 1917; La pression artérielle dans les branches de l'artère centrale de la rétine: Nouvelle technique pour la déterminer, ibid. **154**:648-666, 1917; La circulation rétinienne à l'état normal et à l'état pathologique: Procédés modernes d'examen et de diagnostic, Paris, Gaston Doin, 1923; The Retinal Circulation in the Normal and Pathologic State, translated by J. E. Lebensohn, Chicago, The Professional Press, Inc., 1928; Quelques considérations sur la pression dans la veine centrale de la rétine, Ann. d'ocul. **168**:513-539 (July) 1931; L'hypertension artérielle rétinienne, Tr. Internat. Cong. Ophth. (1937) **1**:87-142, 1938. Bailliart, P.; Magniel, and Saragea: Messures de la pression artérielle rétinienne et de la tension céphalorachidienne dans quelques cas d'hypertension artérielle, Arch. d. mal. du cœur **17**:289-294 (May) 1924.

34. Suganuma, S.: Studien über den Blutdruck in der Zentralarterie der Netzhaut: I. Ueber den Blutdruck in der Zentralarterie der Netzhaut bei gesunden Menschen und über seine Beziehung zum allgemeinen Blutdruck, Klin. Monatsbl. f. Augenh. **96**:74-84 (Jan.) 1936; II. Ueber den Blutdruck in der Zentralarterie der Netzhaut bei verschiedenen Formen von allgemeiner Hypertonie und über die sog. isolierte zephale Hypertension, ibid. **97**:498-514 (Oct.) 1936; III. Ueber den Blutdruck in der Netzhautarterie während des Verlaufes der normalen Schwangerschaft und der Schwangerschaftstoxikose, sowie über seine frühdiagnostische Bedeutung, ibid. **99**:637-654 (Nov.) 1937. Koch.[11]

35. Bright, R.: Cases and Observations Illustrative of Renal Disease Accompanied with the Secretion of Albuminous Urine, Guy's Hosp. Rep. **1**:338-379, 1836; Tabular View of the Morbid Appearances in One Hundred Cases Connected with Albuminous Urine, ibid. **1**:380-400, 1836.

36. Johnson, G.: I. On Certain Points in the Anatomy and Pathology of Bright's Disease of the Kidney; II. On the Influence of the Minute Blood-Vessels upon the Circulation, Med.-Chir. Tr. **51**:76, 1868.

37. Gull, W. W., and Sutton, H. G.: On the Pathology of the Morbid State Commonly Called Chronic Bright's Disease with Contracted Kidney, Med.-Chir. Tr. **55**:273-326, 1872.

38. Keith, N. M.: General Consideration of the Relationship of Hypertension to Renal Disease, Proc. Staff Meet., Mayo Clin. **15**:465-467 (July 24) 1940.

39. Mahomed, F. A.: Some of the Clinical Aspects of Chronic Bright's Disease, Guy's Hosp. Rep. **24**:363-426, 1879.

disease because he felt that these persons would have chronic Bright's disease sooner or later. In 1893 von Basch [40] stated: "There are numerous cases in which examination reveals a high tension of the pulse but the older characteristics of outspoken arteriosclerosis are either absent or minimal." He termed this condition "latent arteriosclerosis." A similar entity was represented by Huchard's [41] "presclerosis," described in 1899.

A modified point of view was advanced by Jores [42] in 1904, who expressed the opinion that the vascular changes in the small arteries that he observed in varying degrees in the viscera in cases of nephritis, but never in the heart and the skeletal muscles, resulted, when present, in materially influencing the progress and outcome of the disease.

The term "hyperpiesia" was introduced by Clifford Allbutt,[43] to whom Wagener and Keith [32] have stated should go "perhaps the greatest credit for the recognition of the syndrome of essential hypertension." Hyperpiesia, atherosclerosis and chronic nephritis were well established entities in 1915, when Allbutt's book, "Diseases of the Arteries, Including Angina Pectoris," was published.

It was Janeway,[44] in 1913, who, in reviewing and summarizing to date the then existing knowledge of cardiovascular-renal disease, spoke of alterations in the vasomotor regulation of the small arterial vessels in essential hypertension, stating:

> The renal disease back of what we call chronic interstitial nephritis is a disease of the small blood vessels and the lesions of the kidney are secondary manifestations. . . . From the standpoint of physiology the high blood pressure is the evidence of arteriolar disease rather than renal. It must be interpreted as a sign of abnormal irritability of the constrictor mechanism. . . . Disturbed vasomotor regulation not permanent obliteration is the usual important factor.

And, in writing of hypertension subsequent to renal disease, he added that hypertension also may develop "in primary irritability of the vasoconstricting mechanism from unkown, probably external causes, which lead eventually to arteriolar sclerosis."[44]

The investigations of Volhard,[45] whose contributions with regard to bilateral hematogenous renal disease were reviewed recently by Wagener and Keith,[32] probably have been the most valuable of recent years. Volhard seems to have been the first [46] (1918) to direct attention to the significance of angiospasm of the arterioles in hypertensive disease. The presence of arterial constriction or hypertonus, however, already had been suspected or recognized, as has previously been noted. Thiel,[47] in the main, expressed agreement with the views of Volhard and accepted the results of Badtke with regard to diameters of retinal vessels in normal and in hypertensive patients. He expressed the opinion that the really alarming sign in cases of pale hypertension is the appearance or onset of angiospasm.

Both Volhard [46] and Goldblatt and his coworkers [48] stated the opinion that the direct action of a vasopressor substance on the walls of the arterioles produces contraction of those vessels. This assumption that prolonged spasm of the arteries or arterioles plays a role in the pathogenesis of hypertension has given rise to considerable discussion. The organic changes in the retinal arterioles subsequent to anigospastic activity in hypertensive disease and in nephritis appear to be more logically explained by Volhard's conception of angiospasm than by any other theory advanced. Wagener [49] reviewed the factors involved in the significance and production of spasm in retinal arteriolar disease and

40. von Basch, S. S. K.: Allegemeine Physiologie und Pathologie des Kreislaufs, Vienna, A. Hölder, 1892.

41. Huchard, H.: Traité clinique des maladies du cœur et de l'aorte, ed. 3, Paris, O. Doin, 1899, vol. 3.

42. Jores, L.: Ueber die Arteriosklerose der kleinen Organarterien und ihre Beziehungen zur Nephritis, Virchows Arch. f. path. Anat. **178**:376-406 (Dec.) 1904.

43. Allbutt, T. C.: Senile Plethora or High Arterial Pressure in Elderly Persons, Tr. Hunterian Soc., 1896, pp. 38-57.

44. Janeway, T. C.: Nephritic Hypertension: Clinical and Experimental Studies, Am. J. M. Sc. **145**:625-656 (May) 1913.

45. Volhard, F.: Elevated Blood Pressure, in Berglund, H., and Medes, G.: The Kidney in Health and Disease, Philadelphia, Lea & Febiger, 1935, pp. 386-416.

46. Volhard, F.: Die doppelseitigen hämatogenen Nierenerkrankungen (Bright'sche Krankheit), Berlin, Julius Springer, 1918.

47. Thiel, R.: Die Bedeutung der Augenuntersuchung für die Diagnose und Differentialdiagnose der Hochdruck- und Nierenkrankheiten, Tr. Internat. Cong. Ophth. (1937) **2**:201-209, 1938.

48. Goldblatt, H.; Lynch, J.; Hanzal, R. F., and Summerville, W. W.: Studies on Experimental Hypertension: I. The Production of Persistent Elevation of Systolic Blood Pressure by Means of Renal Ischemia, J. Exper. Med. **59**:347-379 (March) 1934. Goldblatt, H.: The Pathogenesis of Experimental Hypertension Produced by Renal Ischemia, J. A. M. A. **108**:675 (Feb. 20) 1937.

49. Wagener, H. P.: (*a*) The Significance of Spasm in Retinal Arteriolar Disease and in Retinitis, Tr. Pacific Coast Oto-Ophth. Soc. **24**:165-177, 1939; (*b*) Retinal Lesions in Nephritis and Hypertension, in Berglund, H., and Medes, G.: The Kidney in Health and Disease, Philadelphia, Lea & Febiger, 1935, pp. 622-631; (*c*) footnote 29.

in retinitis and considered the theories advanced in explanation of Volhard,[45] Mylius,[50] Friedenwald,[28] Koyanagi,[51] Fritz,[52] Pal[53] and others.

Pal's interpretation of the nature of arterial narrowing denies the possibility of the existence of continuous spasm, since it is not in accord with biologic experience with smooth muscle and with histologic investigations. Spasm, while a normal physiologic function in lower animal forms, also serves useful purposes in the higher animals. It frequently, however, is an untoward phenomenon in civilized man, particularly when the external environment becomes unduly competitive. It might be assumed that spasm as a protective function in urban man, therefore, often is harmful when it occurs as a physiologic response to stimuli originating in his now less dangerous environment. This response then becomes at first, certainly, one that is physiologic, but abnormally so, to the point where it might be considered psychopathophysiologic. Pal, who was one of the chief exponents of the existence of arteriolar spasm, stated the belief that the generalized arteriolar narrowing thought to be present with pathologic elevations of systemic blood pressure is due to continuous hypertonicity and not to active vascular spasm as such.

The resistance to the flow of blood in the arterioles is dependent on three factors: the viscosity of the blood, the velocity of the stream and the size of the lumen. The velocity of flow is much greater in the arterioles than it is in the capillaries normally, and the size of the lumen is determined, within limits, by the tone of the vessel. The tone of the arterioles constantly is under the control of the vasomotor center. This center is in the floor of the fourth ventricle, at the level of the apex of the calamus scriptorius. The thoracic and the upper two lumbar segments of the spinal cord are supplied by fibers which pass down the cord from this center. These fibers end in the gray matter of the lateral horn in connector cells of the sympathetic system.

Vasoconstrictor fibers of the sympathetic nerves arise from these connector cells. Some vasodilator fibers are found in these sympathetic nerves, as well as in the posterior nerve roots. Sympathetic vasomotor nerves have been demonstrated in nearly all organs and tissues, including, more recently, the pia mater and the substance of the cortex of the brain (Cobb and Talbott,[54] Forbes[55] and Forbes and Wolff[56]). Vasoconstrictor nerves are supplied to the eye through the cervical sympathetic nerves; however, no vasodilator fibers have been demonstrated anatomically in the eye, although the sympathetic fibers can be followed, through the nerve of Tiedemann, to the retinal vessels and to the uveal vessels (Duke-Elder[26]). Langley and Anderson[57] and Henderson and Starling[58] have proved the constricting effect of these nerves on the uveal vessels. The choroidal vasculature comprises approximately 80 per cent of the intraocular circulation (Duke-Elder[26]). Duke-Elder[59] suggested that the capillary bed of the choroid constricts to lower that intraocular tension when increased systemic arterial pressure has been effected by large doses of solution of epinephrine hydrochloride (1:1,000).

Vasoconstricting and vasodilating activities of the retinal vessels usually have been investigated together. It is not definitely established whether there is increased peripheral resistance in diffuse arteriolar disease with hypertension. The plethysmographic investigations of Prinzmetal and Wilson[60] and Pickering[61] indicate that, while constriction of the peripheral arterioles does occur, the constricting mechanism is independent of the vasomotor nervous system, since anesthetization of the nerves of the latter does not result in

50. Mylius, K.: Funktionelle Veränderungen am Gefässsystem der Netzhaut, Berlin, S. Karger, 1929.

51. Koyanagi, Y.: Veränderungen auf der Netzhaut bei Hochdruck: Pathologische Anatomie, Tr. Internat. Cong. Ophth. (1937) **1**:145-283, 1938.

52. Fritz, M.: Rapport des pressions sanguines humérales et rétiniennes, Bull. Soc. belge d'opht. **67**:67-75, 1934.

53. Pal, J.: Ueber die kinetische und die tonische Gefässverengerung, Med. Klin. **25**:702 (May 3) 1929.

54. Cobb, S., and Talbott, J. H.: Studies in Cerebral Circulation: II. Quantitative Study of Cerebral Capillaries, Tr. A. Am. Physicians **42**:255-262, 1927.

55. Forbes, H. S.: The Cerebral Circulation: I. Observation and Measurement of Pial Vessels, Arch. Neurol. & Psychiat. **19**:751-761 (May) 1928.

56. Forbes, H. S., and Wolff, H. G.: The Cerebral Circulation: III. The Vasomotor Control of Cerebral Vessels, Arch. Neurol. & Psychiat. **19**:1057-1086 (June) 1928.

57. Langley, J. N., and Anderson, H. K.: On the Mechanism of the Movements of the Iris, J. Physiol. **13**: 554-597, 1892.

58. Henderson, E. E., and Starling, E. H.: The Influence of Changes in the Intra-Ocular Circulation on the Intra-Ocular Pressure, J. Physiol. **31**:305-319 (Aug. 22) 1904.

59. Duke-Elder, W. S.: Recent Advances in Ophthalmology, Philadelphia, P. Blakiston's Son & Co., 1934, p. 115.

60. Prinzmetal, M., and Wilson, C.: The Nature of the Peripheral Resistance in Arterial Hypertension with Special Reference to the Vasomotor System, J. Clin. Investigation **15**:63-83 (Jan.) 1936.

61. Pickering, G. W.: The Problem of High Blood Pressure in Man, Proc. Staff Meet., Mayo Clin. **14**: 310-320 (May 17) 1939.

vasodilatation. Cusick,[62] in 1939, reviewed this field of investigation in his study of the changes in the caliber of the retinal arterioles of patients during an induced vasospastic reaction. He concluded with Herrel [13] that transitory reductions in caliber of the retinal arterioles occur with general vasopressor reactions to cold and tobacco in certain subjects. Contradictory observations have been reported, but, in the main, experimental evidence and clinical experience appear to be in agreement that there exist vasoconstrictor fibers to the retinal vessels and that vasodilater fibers probably also are present. The recent observations of Cusick, Benson and Boothby,[25b] however, suggest that the retina also possesses an autoregulatory mechanism for the control of its circulation.

Wagener,[12] in 1931, was able to demonstrate a measurable dilatation of the retinal arterioles on postoperative examination in 75 per cent of the eyes of 37 patients in whom the inferior cervical and the first and second thoracic sympathetic ganglions and their intervening trunks had been removed. The veins in 52 per cent of the eyes were found to be dilated, but it was noted that the retinal vessels tended to regain their tone within one year after the operation. It is of significance in this connection that notably better results were reported by Wagener, Cusick and Craig [63] in the surgical treatment of hypertensive disease in the patients whose retinal arterioles lacked structural change than in patients with advanced retinal arteriosclerosis. Lobeck,[20] in 1937 was able to demonstrate measurable dilatation of retinal arterioles immediately subsequent to the subcutaneous injection of acetylcholine, choline and theophylline ethylenediamine in human subjects. Recently, I have observed appreciable (measurable) dilatation of the retinal arterioles in man subsequent to administration of theophylline ethylenediamine following coronary anginal pain induced by breathing a mixture of low oxygen concentration. The vessels were observed prior to inhalation of the gas, and narrowing was seen. The administration of the drug immediately was followed by smooth, generalized dilation to a diameter slightly greater than that observed before the gas was inhaled. This change was transient.

Contraction of the ocular arterioles usually accompanies rises in systemic blood pressure, causing a decrease in the total flow of blood through the eye and a fall in capillary pressure. Thus, there occurs no increase in the intraocular tension. Duke-Elder [26] stated that there is less pronounced vasoconstriction of the arterioles of the eye than of those elsewhere in the body when elevation of the blood pressure is brought about by posterior pituitary injection U. S. P. and that the increase in blood pressure, therefore, is transmitted to the capillary bed, with a resultant passive following of the blood pressure by the intraocular tension.

In general, the normal systemic blood pressure is maintained by the adjustment of the capacity of the peripheral vascular bed to the volume of the circulating blood. The latter is a constant, and the former is a variable. The vasomotor center, by controlling the activity of the arterioles in the unaffected parts, compensates for the local variations in vascular capacity.

Wagener and Keith,[32] in discussing the mechanism involved in the production of pathologic elevation of blood pressure, stated:

> In any discussion of the abnormal physiologic processes which may give rise to hypertension, four obvious factors must be considered; namely, (1) increased cardiac output, (2) increased viscosity, (3) increased total blood volume and (4) increased resistance in the peripheral circulation.

The increase of resistance to the flow of blood in the peripheral circulation in cases of systemic arterial hypertension, according to Pal,[53] is the result of widespread, continuous hypertonus. Prinzmetal and Wilson,[60] among others, supported the conception of a generalized vasoconstriction of the small arteries and arterioles associated with essential hypertension and with chronic nephritis. Wagener [49a] stated the opinion that this is a functional narrowing of the lumen of the arterioles, at least in the earlier phases of the development of the pathologically elevated blood pressure, and that structural changes in the walls of these small arterial vessels are not demonstrable during this period. The reduction in caliber of the retinal arterioles in this condition of continuous hypertonus may be observed with the ophthalmoscope as essentially a smooth, generalized narrowing, which may differ in degree in the several vessels under observation but which will be uniform throughout the course of a particular arteriole.

The observations of Cusick and Herrel [13] suggest that this smooth narrowing may be considered to be due to an increase in vasomotor tone, probably brought about by excessive sympathetic vasoconstrictor stimulation. That the arteriolar contraction, on the other hand, may

62. Cusick, P. L.: Changes in the Retinal Arterioles of Patients During an Induced Vasospastic Reaction, Thesis, University of Minnesota Graduate School, 1939.

63. Wagener, H. P.; Cusick, P. L., and Craig, W. M.: The Retina in Surgical Cases of Primary Hypertension, Tr. Am. Ophth. Soc. **37**:379-394, 1939.

be the result of a direct action of a vasopressor substance on the walls of these vessels is the view expressed by Volhard,[64] by Goldblatt and his followers [48] and by Pickering.[61] It has been demonstrated ophthalmoscopically by Lobeck,[20] Neame,[14] Badtke [22] and others that, however the constriction is produced, the retinal arterioles become generally narrowed fairly proportionally to the severity of the hypertensive disease and, presumably, to the increased elevation of the systemic blood pressure.

The persistence of elevation of blood pressure in persons with diffuse vascular disease results in the production of ophthalmoscopic signs which probably are indicative of the development of hypertrophy of the media of the arterioles. This is the most common histologically observed structural change in the arteriolar walls of persons with chronic hypertensive disease. Sclerosis of these walls of the medial or of the intimal type ultimately will ensue, and that its development probably does have some effect in narrowing the lumen of the arterioles in hypertensive persons is suggested by certain observations in my study of the retina in cases of hypertension, in 1941.[11]

The end stages of hypertensive disease and of nephritis are essentially similar, although they may be reached through various primary modes of development, as Wagener and Keith [32] have stated. The same basic processes logically may be assumed to be concerned in the production of the retinal and of the renal complications. The ophthalmoscopic differentiation of the retinal changes of hypertension from those of nephritis probably lies in the sequential development of the various factors comprising the terminal picture rather than in the final histologic picture. Efforts to differentiate clinically these various systemic vascular diseases have stimulated ophthalmoscopic study of the retina and of the retinal vascular lesions as an aid to diagnosis, and the presence of such signs may also be of prognostic and therapeutic value.

Gowers,[65] in 1876, in studying the retinal vessels ophthalmoscopically in the absence of retinitis, appears to have been the first to employ the ophthalmoscope as an aid in diagnosis of systemic vascular disease. He wrote:

> . . . When in chronic Bright's disease, the pulse is incompressible, there may as a rule be seen reduction in size of the retinal arteries independently of any retinal disease; and this reduction in size is fairly proportionate to the increased arterial tension.

64. Volhard (footnotes 45 and 46).

65. Gowers, W. R.: The State of the Arteries in Bright's Disease, Brit. M. J. **2**:743-745 (Dec. 9) 1876.

Gowers apparently was the first to express the opinion that the reduced caliber of the retinal arteries was due to constriction of the vessels, when he stated:

> When the retina is free from local disease, there is no reason to believe that the retinal artery and vein differ in their condition from other arteries and veins of the same size, and, therefore, any marked change in their state apart from cerebral or ocular disease may be taken as evidence of a similar change throughout the vascular system. . . .
>
> There is, of course, nothing in the fact that the retinal arteries are small in Bright's disease; it has long been remarked as a common feature in albuminuric retinitis . . . but it is usually regarded as a consequence of the retinal change, and the points on which I would insist are that it occurs also quite independently of the retinal change and stands commonly in direct relation to another condition, the blood tension.

Wagener and Keith [32] stated: "In essence, these statements cover the recognized significance of retinal vascular lesions in systemic disease today." The relation of the retinal lesions to general vascular disease and the significance of the ophthalmoscopic signs of arteriosclerosis were studied and interpreted by Raehlmann,[66] Gunn [67] and Röhmer [68] in 1889, 1892 and 1906, respectively, and have been investigated by others since then. The concept of retinal arteriosclerosis as a part of general and cerebral arteriosclerosis grew in concept, and it has become increasingly accepted in recent years "that the lesions observed ophthalmoscopically in the retinal arterial branches are really arteriosclerotic in their characteristics and systemic signfications." [32] O'Hare and Walker,[69] in 1924, pointed out that, almost without exception, so-called retinal arteriosclerosis is a part of hypertensive disease and diffuse arteriolosclerosis and not of atherosclerosis. Parenthetically, it was O'Hare,[70] in 1920, who pointed out the marked lability of the systemic blood pressures in moderately advanced hypertensive disease.

66. Raehlmann, E.: Ueber ophthalmoskopisch sichtbare Erkrankung der Netzhautgefässe bei allgemeiner Arteriosklerose, mit besonderer Berücksichtigung der Sklerose der Hirngefässe, Ztschr. f. klin. Med. **16**:606-654, 1889.

67. Gunn, R. M.: Ophthalmoscopic Evidence of (1) Arterial Changes Associated with Chronic and Renal Disease, and (2) of Increased Arterial Tension, Tr. Ophth. Soc. U. Kingdom **12**:124-125, 1892; On Ophthalmoscopic Evidence of General Arterial Disease, ibid. **18**:356-381, 1898.

68. Röhmer, J.: Rapport sur l'artério-sclérose oculaire, Paris, G. Steinheil, 1906.

69. O'Hare, J. P., and Walker, W. G.: Arteriosclerosis and Hypertension, Arch. Int. Med. **33**:343-349 (March) 1924.

70. O'Hare, J. P.: Vascular Reactions in Vascular Hypertension, Am. J. M. Sc. **159**:369-380 (March) 1920.

Wagener and Keith, together with their co-workers and followers, in the past two decades have evolved a logical and useful clinical classification of hypertensive disease in the absence of an established etiology. These workers began their joint studies in 1920 with the clinical recognition of a case of so-called malignant hypertension. The patient had been admitted to the hospital with a diagnosis of chronic glomerulonephritis. Typical "albuminuric retinitis" was present, but renal function was far too good for a patient with terminal chronic diffuse nephritis. These workers, by further clinical, ophthalmologic and pathologic studies, identified a syndrome characterized by hypertension and diffuse arteriolar changes throughout the body. A small series of cases of this syndrome was presented in 1924,[71] and Keith, Wagener and Kernohan,[72] in 1928, reported a series of 81 cases in greater detail. In many of these cases the diagnosis frequently had been made prior to the development of serious impairment of the cardiac, renal, cerebral and retinal functions, and it was in this respect that the condition differed from the *bösartig* hypertension of Volhard [45] and the malignant nephrosclerosis of Fahr.[73] The retinal changes in the cases reported by Keith and by Wagener, individually or as co-authors, differed in degree from one patient to another, but certain distinctive changes always were present. The disease rapidly progressed, and, in their 1928 series, 78 per cent of the patients had died within a period of one year.

Another group of cases of hypertensive disease gradually came to be recognized in which the course was less rapid, remissions sometimes occurred and there existed mild vasospastic retinitis. The retinitis always was an important diagnostic feature. There came to be recognized also, a group in which marked hypertension existed without a demonstrable break in the functions mentioned but in which there was present more advanced narrowing and sclerosis of the retinal arterioles than in the cases of truly "benign," "simple" hyperpiesia. The authors preferred to designate these four groups by numbers rather than apply to them confusing descriptive terms. Thus, they termed as group 1 the cases of the usual, benign form; as group 2, cases in which there was more marked hypertension but few untoward symptoms and no retinitis; as group 3, the cases in which mild vasospastic retinitis existed and as group 4, the cases in which the syndrome of so-called malignant hypertension was present.[74] It is of interest to mention here that cases of chronic glomerulonephritis originally were designated as group 1 by Keith, Barker and Kernohan,[75] in 1931, and that the present group 4 was called group 5. The present classification of primary, or essential, hypertension into four groups was first used by Keith, in 1932.[76] Cases of acute vasospastic hypertension was added to this group of hypertension in the exhibit offered by Keith and Wagener in 1934 (personal communication).

Keith [76] applied the prefixing phrase of diffuse arteriolar disease with hypertension to the numerical groupings in 1932, and these authors and their co-workers and followers have continued to employ this clinical terminology and classification in the diagnosis of hypertensive disease. The degree of narrowing and sclerosis of the retinal arterioles, as observed with the ophthalmoscope by these workers, also is graded from 1 to 4, depending on the intensity of the changes seen.

Wagener and Keith [32] stated:

> Clinical differentiation of the four groups is usually not difficult. Patients of groups 1 and 2 necessarily must have good retinal, cerebral, cardiac and renal function, whether the hypertension be labile, or high, with a tendency to fixation. In cases of group 3 there is evidence of dysfunction of one or of several of the organs belonging to the arterial system, while in group 4 these functional disturbances become more definite and final failure may occur because of a simultaneous serious interference in the blood supply to the retina, brain, heart and kidneys. A given case may be markedly progressive and pass from group 1 to group 4 in a short period. On the other hand, there are cases in which there is little progression over a period of many years; the majority of these belong to group 1 but a moderate number to group 2 and a few to group 3. Because the arterioles throughout the organs of the body seem to be the chief point of attack and can be readily visualized only in the retina, the findings obtained by the ophthalmoscope are very important.

A further report on an increased number of cases of essential hypertension recently has been

71. Wagener, H. P., and Keith, N. M.: Cases of Marked Hypertension, Adequate Renal Function and Neuroretinitis, Arch. Int. Med. **34**:374-387 (Sept.) 1924.

72. Keith, N. M.; Wagener, H. P., and Kernohan, J. W.: The Syndrome of Malignant Hypertension, Arch. Int.. Med. **41**:141-188 (Feb.) 1928.

73. Fahr, T.: Ueber Nephrosklerose, Virchows Arch. f. pa h. Anat. **226**:119-178 (June) 1919.

74. Wagener, H. P.: The Clinical Interpretation of Retinal Vascular Lesions in Hypertension and Nephritis, Pennsylvania M. J. **40**:705-711 (June) 1937.

75. Keith, N. M.; Barker, N. W., and Kernohan, J. W.: Histologic Studies of the Arterioles in Various Types of Hypertension, Tr. A. Am. Physicians **46**: 66-68, 1931.

76. Keith, N. M.: II. Cardiovascular Diseases in Relation to the Retina, Tr. Am. Acad. Ophth. **37**: 37-49, 1932.

published by Keith, Wagener and Barker.[77] The classification just mentioned was employed and conclusions were drawn with regard to the prognosis in 219 cases, representing all four groups. An actuarial survival curve reveals that there is a death rate of 10 to 12 per cent within a period of one year following the time of the first examination in groups 1 and 2. This is in contrast to the 35 per cent mortality in group 3 and the 79 per cent in group 4 in the same length of time.

COMMENT

"Normal blood pressure" in the human being is somewhat difficult of accurate definition in terms of millimeters of mercury. From his statistical study of human blood pressures in 2,282 men and 3,258 women, Wetherby[78] reached the conclusion that it was impossible to draw a definite line between normal and elevated or depressed pressures on the basis of single readings. Variations in pressures in states of health occur within narrow limits under conditions of exercise, emotion and similar stresses. Blood pressures which consistently are higher than 150 mm. of mercury systolic and 90 mm. of mercury diastolic usually are interpreted as indicating the existence of pathologic hypertension regardless of the age of the patient. These figures for the upper limits of normal clinical blood pressures in the human being are those accepted by most workers in this field.

Blood pressure, as Mountain and Allen[79] recently have reemphasized, is not a static, but a labile, manifestation of physiologic activity of the human body. These authors concluded, in their studies on the fluctuation of blood pressure in patients with essential hypertension, that the pressure varies widely in the individual cases and that the greatest fluctuation occurs in groups 2 and 3 because of the pronounced elevation of the blood pressure with relatively little fixation of it in these groups. Since, as is known, normal blood pressure fluctuates only within rather narrow limits in most cases, these authors found comparatively slight variations in group 1 because the elevation of the systemic pressure was not great. Similarly, in cases of hypertension, group 4, the fluctuation also was slight, but only because the systemic pressure was relatively fixed. They stated the opinion that the soundness of grouping cases of hypertension on the basis of retinal changes is confirmed by the existence of progressive elevation of both the maximal and the minimal brachial systolic and diastolic blood pressures in each of the four successive groups of hypertension classified according to the criteria of Keith and Wagener.

Values for similar fluctuations of the brachial and of the retinal diastolic blood pressures were

TABLE 4.—*Mean Caliber of Retinal Arterioles in Microns as Measured by Various Authors*

Keith Wagener[32] Classification	Volhard[45] Classification	Lo Cascio[18]	Wagener[12]	Neame[14]	Lobeck[20]	Kühn[21]	Badtke[22]		Koch[11]	
							Range	Mean	Range	Mean
Normal blood pressure	Normal blood pressure	110 to 120	50 to 120	86 to 94	88 to 150	86 to 138	90 to 112	105	47 to 140	91
Group 1	"Red" hypertension	.....	.....	.....	74 to 127	...	100 to 145	117	46 to 124	77
Group 2	Transition forms	.....	.....	.....	...	...	60 to 85	70	31 to 111	67
Group 3	"Pale" hypertension	.....	.....	.....	...	...	...	...	31 to 111	60
	Genuine nephrosclerosis	.....	.....	.....	27 to 58	...	...	...		
	Primarily contracted kidney	.....	.....	.....	...	...	60 to 91	70		
Group 4	Genuine nephrosclerosis with albuminuric retinitis	.....	.....	.....	26 to 33	...	...	...	31 to 94	41
	Malignant hypertension	.....	.....	.....	53 to 81	...	...	...		
Chronic glomerulonephritis with normal brachial diastolic blood pressure		.....	.....	.....	...	...	96 to 125	109	47 to 124	84
Chronic glomerulonephritis with elevated brachial diastolic blood pressure		.....	.....	.....	38 to 73	...	60 to 78	70	37 to 93	60

77. Keith, N. M.; Wagener, H. P., and Barker, N. W.: Some Different Types of Essential Hypertension: Their Course and Prognosis, Am. J. M. Sc. **197:** 332-343 (March) 1939.

78. Wetherby, M.: A Comparison of Blood Pressure in Men and Women: A Statistical Study of 5,540 Individuals, in Berglund, H., and Wedes, G.: The Kidney in Health and Disease, Philadelphia, Lea & Febiger, 1935, pp. 370-386.

79. Mountain, G. E., and Allen, E. V.: Vascular Clinics: XV. Fluctuation of Blood Pressure in Essential Hypertension, Proc. Staff Meet., Mayo Clin. **16:** 260-263 (April 23) 1941.

not sought in the individual cases in my study [11]; however, the fact that variations probably occur is indicated by the range of values obtained with reference to the caliber of the arterioles and the systemic and local blood pressures recorded. Rather wide variations in arteriolar caliber have been reported by others (tables 2 and 4). No previous studies have been made, however, in an attempt to correlate the diameters of the retinal vessels with both the systemic and the local ocular blood pressure, nor, apparently, have the previous investigations been carried out on large numbers of arterioles in sufficiently representative groups of patients. That this may not always be necessary, however, is indicated in these tables with regard to the values reported by other investigators for normal retinal arterioles and without reference to the particular device employed in obtaining these measurements. The mean value has not always been given by each author; however, it will be observed that not only the range but the mean, when given, are in rather general agreement.

There exists only two reports (Lobeck,[20a] Badtke [22]) concerning retinal vascular diameters in cases of hypertensive disease, and these are somewhat difficult to evaluate with reference to my study [11] because the authors employed the classification of Volhard.[45] In the main, however, as will be observed in table 4, the values given by Lobeck [20] and by Badtke [22] correspond to those I obtained, although these values do not approximate each other quite as well as do the figures given by the various workers for normal values.

The mean caliber of the retinal arterioles, as well as the range of calibers, in my study generally is more in accord with the values given by Lobeck than with those obtained by Badtke, although both these workers used the heliometer apparatus devised by the former. No effort was made in my study to differentiate "red" and "pale" hypertension as such or to distinguish specifically the "transition forms." Since it is believed, with Wagener and Keith, that an angiospastic tendency is present in practically all cases of hypertension, it is difficult, on the basis of vascular physiology, to correlate dilatation of the arterioles with a moderate increase in brachial systolic blood pressure even in the presence of a normal systemic diastolic blood pressure.

Badtke obtained a mean retinal arteriolar caliber of 117 microns for 56 patients with "red" hypertension. This figure represented a range of 100 to 145 microns, and, for the most part, he measured only the superior temporal arterial vessels at the margin of the disk. His figures for "red" hypertension, as will be seen in table 4, are greater than those obtained in his series of 25 normal patients; otherwise the values obtained by Lobeck and by Badtke in cases of the various forms of hypertensive and nephritic disease appear to be in general agreement with mine.[11]

It is possible that Badtke, with reference to "red" hypertension, encountered a group of patients who might better have been classified as having diffuse arterial disease with atherosclerosis, but he did not give the actual figures for the systemic blood pressures in this group, of 56 patients. It might be reasonable to suppose that relative retinal vascular widening in relatively elderly patients may accompany increases in brachial systolic blood pressures, particularly if the elasticity of the larger arteries had been diminished. It is possible, also, that Fritz [52] encountered a similar group of patients, after the study of which he advanced, among others, the theory that certain vascular regions exist in which the arterioles fail to contract to a sufficient degree. He expressed the opinion that the blood thus will enter the capillaries under too great a pressure and that this would permit hemorrhagic and serous transudation through the capillary walls. He also assumed that there might be a more or less uniform degree of arteriolar attenuation in some stages of hypertensive disease and that this would permit uniform optimal perfusion of the retinal capillaries without the development of retinitis. The arterioles are constricted unequally in other stages, he thought, so that the blood in the capillaries would enter under too low a pressure and result in infarction. Fritz supported this hemodynamic or mechanical theory by numerous observations on the retinal pulse and the intraocular blood pressure reactions. His hypotheses have not yet been proved.

60 East Seventy-Fifth Street.

News and Notes

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

Research Study Club of Los Angeles.—The Research Study Club of Los Angeles announces its fifteenth annual mid-winter postgraduate clinical assembly in ophthalmology and otolaryngology, Jan. 21 to Feb. 1, 1946, and special courses in applied anatomy and cadaver surgery of the head and neck, February 1 to 5. Guest speakers for ophthalmology and their subjects are: Dr. Alan C. Woods, immunology in ocular disease; Dr. Jack S. Guyton, newer methods of surgical approach in ophthalmic surgery; Dr. Meyer Wiener, demonstrations in surgery of the eye; Dr. Frederick C. Cordes, recent advances in ophthalmology, and Mr. Irving B. Lueck, analysis of various optical problems, with special reference to lenses. Dr. Herbert M. Evans, professor of biology at the University of California, at Berkeley, Calif., will discuss recent advances in nutrition in relation to problems of the eye, ear, nose and throat, with particular reference to nicotinic acid and riboflavin deficiencies in man.

Applications should be made to Pierre Viole, M.D., 1930 Wilshire Boulevard, Los Angeles 5.

Research Project in Cause of Blindness Due to Onchocerciasis in Guatemala.—Dr. William B. Clark, professor of ophthalmology, Tulane University of Louisiana School of Medicine, has just returned from Yepocapa, Guatemala, where he spent six weeks instituting a research project in the cause of blindness in patients with onchocerciasis in Guatemala. This project is sponsored jointly by the Caribbean Sector of the Pan American Sanitary Bureau and the Department of Health of Guatemala. The project is being continued in Dr. Clark's absence by Dr. Bertha Riveroll Noble, of México, D.F., who was a former Pan American Kellogg Foundation Fellow in Ophthalmology at Tulane University from 1943 to 1945. Dr. Clark will return to Guatemala in January 1946, to close out or continue the project, as the results of the investigation justify.

Pan-American Congress of Ophthalmology.—The Pan American Airways, Inc., is offering a 15 per cent reduction on fares to delegates attending the Pan-American Congress of Ophthalmology at Montevideo, Uruguay.

The fare from Miami to Montevideo is $486 one way or $874.80 round trip, if traveling via the east coast; and $504 one way or $907.20 round trip, if traveling via the west coast.

For any delegates who may be interested in making a circle trip around South America the fare will be $891.

All these fares are subject to 15 per cent United States transportation tax. However, as already mentioned, these fares are also subject to 15 per cent discount.

"Lectures on Motor Anomalies."—A third printing of Alfred Bielschowsky's "Lectures on Motor Anomalies" is now on sale at Dartmouth College Publications, Hanover, N. H., at $1.50 per copy, postpaid.

Obituaries

N. BISHOP HARMAN, M.B.

1869 — 1945

Bishop Harman was born in London and received his education at Cambridge and at Middlesex Hospital, where he qualified in medicine and became a Fellow of the Royal College of Surgeons in 1898. He was one of the first to become interested in the prevention of blindness and in the education of children with defective eyesight after the unrelieved defects of vision were noted in school children. He was instrumental in instituting compulsory notification of cases of ophthalmia neonatorum. Harman was one of the oculists appointed to superintend the regular testing of children's eyesight, and he instituted a number of reforms, such as cessation of sewing by artificial light and the use of slates and pencils. As ophthalmic consultant to the London County Council, he recognized the need for separate classes for children with high myopia. These sight-saving schools were established in London in 1908 and were fitted out with special desks and apparatus. These methods were adopted in the English provincial centers and were followed in the United States. Improved standards of lighting and of school books were devised through Harman's endeavors, and he was one of the main contributors in the diminution of visual defects from preventable causes in childhood.

Harman was president of the section of ophthalmology of the *British Medical Journal* meeting at Winnipeg, in 1930. In addition to writing his excellent "Aid to Ophthalmology," he devised a number of new and ingenious instruments for ophthalmologists. His last interest was in testing night vision, which in the recent war was of such importance. He constructed a simple but practical apparatus for this examination. In his long connection with the *British Medical Journal* he was one of its most active members and served as treasurer for fifteen years.

ARNOLD KNAPP.

Correspondence

MARFAN'S SYNDROME

To the Editor:—The August 1945 issue of the ARCHIVES, page 112, carries a paper entitled "Marfan's Syndrome with Unusual Complications," by Dr. James L. McGraw.

One feels that the title does not harmonize with the contents of the paper; the unusual complications do not have a casual connection with the anomaly but are only postoperative complications of extraction of the lens in a given case of Marfan's syndrome. In the opening paragraph it is stated that Marfan described, in 1896, the case of a young girl with the symptom complex of arachnodactyly and ectopia lentis. The statement is erroneous. In my paper on Marfan's syndrome (ARCH. OPHTH. **27**: 477 [March] 1942), I pointed out that the knowledge of congenital dislocation of the lens and arachnodactyly began with the observation of Marfan, who noted a peculiar and until then undescribed symmetric malformation of the four extremities in a 5½ year old girl. The malformation was characterized by elongation of the bones and a certain degree of attenuation, more pronounced in the distal parts. There were contractures of the fingers, poor musculature, pronounced deficiency of subcutaneous fat and, furthermore, a considerable spur of the os calcis. Marfan described the fingers and toes as "spider-like" and called the syndrome *dolichosténomélie*. In Marfan's case and, similarly, in the other cases of the early literature there is no mention of ocular symptoms. Boerger's report (1914) was the first in the literature to call attention to the presence of the characteristic symptoms of congenital luxation of the lens and iridodonesis with enlarged cornea and hydrophthalmos. Poynton and Maurice demonstrated a case before the Medical Society of London in 1923, and in the discussion following the report "a member of the medical society" spoke of a patient under his care who had the identical syndrome, but with congenital dislocation of the lenses also. The first paper to emphasize the importance of ocular symptoms is that of Ormond and Williams (1924).

Marfan, in 1896, described only the typical habitus of the body; that the congenital dislocation of the lens is part of the syndrome became known only much later and through the observation of other authors.

ANDREW RADOS, M.D., NEWARK, N. J.
31 Lincoln Park (2).

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Congenital Anomalies

A CONTRIBUTION TO THE STUDY OF ANOPHTHALMIA WITH DESCRIPTION OF A CASE. T. ROGALSKI, Brit. J. Ophth. **28:** 429 (Sept.) 1944.

The anomaly was seen in a full term male child who died of miliary tuberculosis of "nearly all the organs" at the age of about 10 weeks. The left side of the mouth was enlarged, and there was a V-shaped cleft pointing to the left orbit and connected with it by a slight groove in the skin. Another groove extended from the right angle of the mouth to the right ear; the right two thirds of the upper jaw, i. e., the part to the right of the harelip, projected beyond the mandible, and the nose slanted downward to the right. The right orbit and eyeball appeared normal, but there was no sign of the left eyeball. No malformations were noted in any other region of the body.

Rogalski suggests that in all cases in which the essential parts of the eye, i. e., the retina proper and the lens, are lacking, whether the mesodermal constituents and the pigment epithelium are present or not, the term anophthalmia should be employed. The term microphthalmia should be reserved for cases in which all the ocular constituents, including the retina proper (or the optic nerve) and the lens, are present either underdeveloped or malformed or only vestigial.

Anophthalmia, thus defined, may be classified into two types: 1. Complete anophthalmia, in which no vestiges of the optic outgrowth can be found. In cases of this type it cannot be determined whether the optic vesicle has been formed and suffered complete atrophy or has never been formed at all. 2. Consecutive anophthalmia, in which an optic vesicle or cup has certainly been formed but has atrophied in the very early stages, leaving, as its vestige, more or less malformed pigment epithelium. Each type may be bilateral or unilateral, and both are almost certainly of genetic origin, the fault being in the neuroblast itself or in other tissues, e. g., in the hematogenic mesoderm, with disturbance of the blood supply.

The author's case belongs to the second type of anophthalmia so defined, and not to the third type described by Ida Mann, as according to her definition it would.

The article is illustrated.

W. ZENTMAYER.

CASUISTIC CONTRIBUTION TO THE MARCUS GUNN SYNDROME. M. ARTIGAS and V. A. VICTORIA, Arch. de oftal. de Buenos Aires **19:** 101 (Feb.) 1944.

Two atypical cases of this syndrome are reported. One patient, a girl 17 years of age, had no ptosis of the superior lid, but when the mandible was lowered the left upper lid would rise. The other patient, a boy 10 years of age, with congenital ptosis of the left upper lid, showed elevation of the ptosed lid on swallowing. In his case the synergism was between the third and the ninth nerve. The article is illustrated.

H. F. CARRASQUILLO.

General Diseases

RUBEOSIS IRIDIS DIABETICA. F. B. FRALICK, Am. J. Ophth. **28:** 123 (Feb.) 1945.

Fralick refers to the work of Salus and Kurz, and notes the interesting features of rubeosis iridis diabetica. He reports 5 cases. One patient, a young person, had severe diabetes with hypertension and extensive damage to the kidneys; 2 patients, elderly, were diabetic and hypertensive; 2 were nondiabetic, both having hypertension and an intraocular melanoma, and 1 had a condition which was diagnosed clinically as an old occlusion of the central retinal vein. The only feature presented by all the patients was the obvious circulatory disturbance, of such a degree that rubeosis iridis and hemorrhagic glaucoma developed. Rubeosis iridis has thus been observed in patients showing rather varied pathologic conditions.

The author cannot support the contention that rubeosis iridis is a manifestation of an ocular syndrome seen in its true form only in diabetic persons. He believes that, given the right pathologic changes in the vascular supply to the eyeball, metabolic abnormalities become established which promote a low grade iritis, with formation of new vessels and secondary peripheral root synechias, resulting eventually in hemorrhagic glaucoma.

W. ZENTMAYER.

OPHTHALMOSCOPY AND THE DIAGNOSIS OF HUMAN ILLNESS. A. J. BEDELL, Am. J. Ophth. **28:** 139 (Feb.) 1945.

In this Jackson Memorial Lecture, before the Colorado Ophthalmological Society, Bedell speaks of Edward Jackson's useful life and enumerates a number of ocular conditions and their importance in relation to general health.

W. S. REESE.

Glaucoma

DEEP-CHAMBER GLAUCOMA DUE TO THE FORMATION OF A CUTICULAR PRODUCT IN THE FILTRATION ANGLE. A. B. REESE, Am. J. Ophth. **27:** 1193 (Nov.) 1944.

Reese gives the following summary:

"The endothelium of the anterior chamber may form a cuticular product at any site where it is normally found. It may also grow and produce this product over sites where it is not normally found; such as, over the trabeculae, across the pupillary area, or along the posterior surface of the iris. When this cuticular product is formed over the inner surface of the trabeculae or in the interstices of the trabeculae, glaucoma may ensue. This may occur as a primary disease with no apparent provocation. It also may occur as a result of inflammation in the anterior chamber, or as the result of trauma to the eye. The endothelial changes occur on the posterior surface of the cornea and permit aqueous to enter the corneal stroma. This gives rise to edema and its sequelae, such as vesicles, bullae, pannus, keratitis, and ulcer. The increased intraocular pressure intensifies these corneal changes. The underlying pathologic change and clinical picture are similar to dystrophia epithelialis corneae on the one hand, and the corneal changes consequent to glaucoma on the other hand, except that in this condition both factors are present and therefore the cornea may dominate the clinical picture."

W. S. REESE.

ROENTGEN IRRADIATION OF CERVICAL SYMPATHETIC TRUNK IN TREATMENT OF GLAUCOMA. J. CASANOVAS, Arch. Soc. oftal. hispano-am. **4:** 594 (July-Aug.) 1944.

After discussing fully this method of treatment, the author concludes that roentgen irradiation of the cervical portion of the sympathetic trunk is useful only as a preliminary measure to prepare for an operation in cases in which the visual field is greatly contracted and the fixation point is menaced. In these cases the field may be improved so as to make the operation possible without endangering the visual acuity. The author further states that the method is useful also as a palliative measure in cases in which there is very high tension and as a preliminary to operative procedures in these cases.

H. F. CARRASQUILLO.

Injuries

TREATMENT OF PERFORATING OCULAR INJURY. L. H. SAVIN, M. Press & Circ. **209:** 134 (March 3) 1943.

After comments on sympathetic ophthalmia and its prevention, indications for early enucleation are given. The composition of an intraocular foreign body is important. The patient may know what metal he was working with, and a sample is often available. Of importance is whether the foreign body is magnetizable or not. Foreign bodies can be accurately localized by localizing roentgenograms, and the author especially mentions the Spaeth ring method. The chemical action of a foreign body is also important. Gold and quartz are inert. Iron and steel cause siderosis. Copper has a chemical action, with early cataractous changes, of which a posterior cortical cataract is significant. Most of the other foreign bodies have slow chemical action. Glass may remain quiet for ten or fifteen years, though sometimes it causes a violent reaction. The effect of organic foreign bodies is also variable. A particle of coal may remain stationary for ten years. Eyelashes may be quiet for many years, unless they form epithelial cysts or pearl tumors. Aluminum alloys were important in World War II, as they are frequently employed in bombs and projectiles. Their chemical effect is under study. Generally they cause mild chemical irritation (iritis, adhesions and lenticular opacities), but necrotic patches have been observed by the author in the retina of man and rabbits. Nonmagnetized tools, steels and cutting tools of tungsten carbide are the present day hazards to industrial workers.

If the foreign body in the interior of the eye is not magnetizable, the question of leaving it alone comes up, as such a body is frequently tolerated for many years. In the case of copper and brass something must be done. An attempt to remove the particle by scleral incision should be made, using a forceps guided by the electric ophthalmoscope. Injured eyes must be kept under observation, as symptoms of sympathetic ophthalmia may develop, with irritability of the injured eye, corneal deposits and an aqueous flare in the second eye. The blood count is of no diagnostic value, as mononuclear leukocytosis is a fallacious guide and is frequently absent in cases of sympathetic ophthalmia. Injections of neoarsphenamine are useful in treatment of sympathetic ophthalmia and are of value as a prophylactic.

ARNOLD KNAPP.

Lens

THE LENS IN ACCOMMODATION. J. G. SINCLAIR, Am. J. Ophth. **28:** 38 (Jan.) 1945.

Sinclair states that after birth, as the lens grows and, at the same time, functions increasingly in accommodation, stresses are set up tangential to the surface, and the (lens) rods become alined in sheets. Between the sheets is albuminous liquid. One zone of stress in particular contains more liquid than the rest and separates the nucleus from the surrounding cortex. Tension at the equator, besides slightly stretching the lens, moves back to the axis on both sides of the nucleus. The nucleus is held

in position by strands that cross the liquid-filled zone, but the whole nucleus is moved forward. The force in this axial expansion is furnished partly through cortical elasticity and partly through hydrostatic pressure, probably based on a small differential in osmotic pressure of lenticular fluid over surrounding fluids. The hydrostatic component operates uniformly in all directions, but its effectiveness on any wall varies directly with the radius of curvature and the thickness of that wall. The curved prismatic cortical rods of the lens tend to become straight in turgor and in doing so supply the necessary elastic component. These forces, together with the fact that the central, or pupillary, zone of the cortex is thin, permit the changes in radius of curvature characteristic of accommodation in this region. This change does not extend to the whole lateral face of the lens.

The lenticular structure here pictured is found in the human eye after birth. In old age the liquid space is reduced or obliterated, and the whole lens becomes like the nucleus, an unyielding and highly refracting mass. The theory of elasticity of the lens presented here does not call for modification of accepted theories of accommodation, but it does make plausible the effectiveness of the force of tension on the ligament of the lens. A diagram shows the mature crystalline lens in tension and in relaxation.

W. ZENTMAYER.

Methods of Examination

PHOTOMETER FOR MEASURING THE SCOTOPIC CANDLEPOWERS OF SELF-LUMINOUS OPHTHALMIC TEST OBJECTS. W. S. STILES, Brit. J. Ophth. **28:** 629 (Dec.) 1944.

A photometer is described with which the scotopic candle power of feeble light sources (down to about 10^{-9} candles) can be measured. Small spots of radium paint, 2 or 3 mm. in diameter and having candle powers in the range covered, are now used (Livingston) to plot the dark-adapted visual field, and the present photometer was designed primarily for their calibration.

A description of the instrument and the conditions of measurement are given.

It may fairly be concluded that, provided the energy distribution of a given test object is roughly matched by choosing a suitable color filter whose computed scotopic transmission is known, an experimental comparison of the test object with the filtered light will give the scotopic value of the test object with satisfactory accuracy, probably to within 12 per cent, except possibly for red test objects. This is true provided the photometric match is made at a scotopic brightness of about 5 by 10^{-4} e.f.c. (4 mm. diameter pupil) and the result is the mean for at least two "normal" observers.

W. ZENTMAYER.

Neurology

VISIBLE RETINAL ARTERIOLAR SPASM ASSOCIATED WITH MULTIPLE SCLEROSIS. R. BRICKNER and C. FRANKLIN, Arch. Neurol. & Psychiat. **51:** 573 (June) 1944.

Abrupt attacks involving various parts of the central nervous system, including the visual apparatus, and usually lasting more than a few minutes or a few hours, are common in cases of multiple sclerosis. However, sudden attacks of one kind or another of brief duration are not uncommon, and the subject of this report is arteriolar spasm associated with such attacks and relief of symptoms with amyl nitrite.

In 2 cases of multiple sclerosis, spasm of the retinal arterioles was coincident with the presence of scotomas. When the scotomas receded or disappeared, the spasm also disappeared; this change followed immediately the inhalation of amyl nitrite in 1 case and was spontaneous in the other. In a third case attacks of blurred vision were promptly relieved by inhalation of amyl nitrite.

R. IRVINE.

ENCEPHALITIS DUE TO TOXOPLASMA. G. SCHWARZ and J. WENDELL, Arch. Neurol. & Psychiat. **52:** 425 (Nov.) 1944.

Two clinical cases of toxoplasma encephalitis of the so-called latent infantile type are presented.

Both patients were white girls 5 years of age. The first child had severe pneumonia at the age of 9 weeks, after which most of her symptoms were first noted. The other child had an illness at birth involving the respiratory tract and congenital dislocation of the hips. Both patients had had convulsive seizures earlier in childhood. The clinical observations were essentially similar. Both patients showed mental deficiency. Visual acuity was greatly reduced, and there was ocular nystagmus. Ophthalmic examination revealed white nerve heads and chorioretinitis, involving particularly the macular areas. Pneumoencephalograms revealed symmetric dilatation of the ventricular systems. Roentgenograms of the skull in each case showed multiple irregular calcifications distributed throughout both cerebral hemispheres, particularly in the occipitoparietal region. A specimen of the blood of the first patient, examined by Dr. Abner Wolf, contained antibodies for Toxoplasma.

R. IRVINE.

Parasites

HISTOLOGIC CHANGES IN AN EYEBALL WITH CYSTICERCOSIS. E. F. PERIS, Arch. Soc. oftal. hispano-am. **4:** 98 (Jan.-Feb.) 1944.

Peris studied an enucleated eye involved in the rare condition of cysticercosis. The eye had been treated with diathermy (electrocoagulation).

Macroscopically, the organ showed beginning phthisis bulbi. Histologically, all the layers and

tissues of the eye except the sclera and the cornea were involved in an intense inflammatory process. Important to note was that the choroid did not show changes similar to those of sympathetic ophthalmia, which other investigators have claimed may arise in the other eye in cases of this disease. The pathologic process in this layer was chiefly that of severe iridocyclitis.

The cestode had undergone calcareous degeneration, as usually happens after its death in the eye.

H. F. CARRASQUILLO.

Physiology

EFFECTS OF STIMULATION AND LESION OF THE MEDIAN LONGITUDINAL FASCICULUS IN THE MONKEY. M. BENDER and E. WEINSTEIN, Arch. Neurol. & Psychiat. **52:** 106 (Aug.) 1944.

Experimental stimulation of the mesial fibers of the median longitudinal fasciculus in the monkey causes ocular adduction. Destruction of these same fibers results in paralysis of adduction and nystagmus in the abducted eye on attempted lateral gaze. This is similar to the ocular syndrome described clinicopathologically in man, known as internuclear paralysis, or ophthalmoplegia internuclearis, which was first described by Bielschowsky in 1902. Convergence and vestibular function are unaffected.

On the basis of these observations, it appears that the ascending fibers in the median longitudinal fasciculus probably represent the terminal portion of the corticobulbar pathway for horizontal conjugate movements of the eyes. This hypothesis has been supported by observations made on a series of stimulations through the reticular substance, the vestibular nuclei and their emerging fibers.

R. IRVINE.

Retina and Optic Nerve

RETINAL DETACHMENT: A SERIES OF 78 CASES IN THE MIDDLE EAST FORCE. H. B. STALLARD, Brit. M. J. **2:** 330 (Sept. 9) 1944.

This is a report of 78 cases of retinal detachment, in 76 of which operation was performed by a combination of surface diathermy placed around the edges of the tear and a few punctures of penetrating diathermy. The interretinal fluid was sucked into a glass tube applied to the sclera over the diathermy penetration. The patients were all soldiers between the ages of 20 and 47. The survey differs from one of retinal detachment seen in civilian practice in that the incidence of myopia was extremely infrequent, namely, in 3 of 78 cases, and injuries peculiar to war were of course absent in civilian practice.

The cases are divided into five groups: (1) cystic degeneration, 13 cases; (2) chorioretinal degeneration, 21 cases; (3) myopia, 3 cases; (4) trauma (civilian), 23 cases, and (5) trauma (military), 18 cases. The results of operation were as follows: (1) cystic degeneration, 13 successes and no failures; (2) chorioretinal degeneration, 21 successes and no failures; (3) myopia, 2 successes and 1 failure; (4) trauma (civilian), 20 successes and 3 failures; (5) trauma (military), 10 successes and 6 failures, giving a total of 66 successes and 10 failures. Any recurrence, however slight, of the retinal detachment was recorded as a failure.

The author then describes more fully the factors which serve as a basis for prognosis. The prognosis is favorable in cases of retinal detachment due to cystic degeneration and to chorioretinal degeneration, especially in men of military age. The prognosis is worse for deep detachments than for shallow ones on account of the distance of the retinal tear from the choroid. In this condition the diathermy needles are passed through the sclera and choroid sufficiently long to reach the retina, but not to perforate it (which is the method advocated by Weve, abstracter). Extensive intraocular degeneration, total detachment, high myopia, very low intraocular pressure and detachment in an aphakic eye are bad prognostic signs. Their presence is least favorable in war injuries. This is explained by the presence of additional trauma from a contusion or a penetrating wound.

ARNOLD KNAPP.

Tumors

SECONDARY CARCINOMA IN THE ANTERIOR CHAMBER. A. J. B. GOLDSMITH, Brit. J. Ophth. **29:** 136 (March) 1945.

A woman aged 64 had undergone a radical mastectomy for carcinoma of the left breast (confirmed by histologic examination) a year previous to the enucleation of the right eye because of secondary glaucoma, suspected to be due to a carcinoma of the choroid. Histologic study showed that the main mass was on the temporal side, where it extended from near the disk as far forward as the equator. In the sclera the growth extended along the canal of one of the ciliary nerves close to the disk. Cells were present in the ciliary body and the iris. In the angle of the anterior chamber malignant cells had burst through the root of the iris and were proliferating into the anterior chamber, where they formed a plaque lying on the surface of the iris, but not intimately connected with it and deriving no blood supply from it. A few cells lay on the posterior surface of the cornea.

The author supplies the following summary:

"A hitherto undescribed condition is described, in which secondary carcinoma is growing in the anterior chamber after the manner of a tissue culture.

"Attention is drawn to the differences in manner of intra-ocular extension between neuro-epithelial retinal tumours and secondary carcinoma within the eye, and it is suggested that

these are largely the result of the different sites in which the two types of growth originate.

"Attention is also briefly drawn to the use made in experimental cancer research of the capacity of the aqueous fluid to act as a tissue culture medium."

W. ZENTMAYER.

Vision

AN INTRODUCTION TO BISHOP BERKELEY'S THEORY OF VISION. M. MURRAY, Brit. J. Ophth. **28:** 600 (Dec.) 1944.

This interesting article does not lend itself to abstraction. The author states and discusses the views of Berkeley, who was an empiricist, the term implying the doctrine that space perception is entirely learnt, as against its being inherited or instinctive. Berkeley has also been described as a tactualist; the term suggests better his doctrine that touch is the space sense *par excellence,* and that sight in its primitive purity is devoid of all space character.

The vista of speculation which the theory opened up formed a background for a controversy which involved many future generations of authorities on visual optics. Two schools of thought exist: the empiricists, of which Helmholtz and Wundt are exponents, and the nativists, which include Hering and Müller. The nativists believe in the theory of inheritance.

W. ZENTMAYER.

SIGHT-SAVING CLASSES. N. BISHOP HARMAN, Brit. M. J. **1:** 53 (Jan. 13) 1945.

The author discusses the technical requirements and potentialities of the school for the partially blind. He gives an extremely interesting historical review of how these sight-saving schools came into being, as he was among one of the earliest persons to become interested in this subject.

In the early work, Harman was much struck by the predominant occurrence of poor sight in girls. Then he found that many of the children in schools for the blind were really not blind; they were highly myopic, could see objects at their own distance very well and mentally were as keen as any other children. This led to the setting up of special classes for the partially blind children, the scheme of work laid down for these classes consisting in (1) oral teaching in groups with the normal children, (2) literary work with the aid of blackboards and chalk and (3) full use of every sort of handicraft which would develop attention, method and skill, with minimum use of the eyes. Special attention was given to such items as proper equipment of the classrooms, exercise books and physical exercises. The most difficult section of the work was manual training; all sewing was prohibited. These classes soon became popular just as they had been popular in the United States, and many of them exist both in London and in the provinces. The important point, the author says, in the training of children with defective vision is proper training so that they use their eyes with intelligence and will be able to carry on with the training after school years.

This article is very interesting; it shows the value of sight-saving classes for the children with partial sight and points out how this training should prevent the loss of sight and save the children from the misery of blindness.

ARNOLD KNAPP.

Society Transactions

EDITED BY DR. W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

THOMAS H. JOHNSON, M.D., *Chairman*

WENDELL L. HUGHES, *Secretary*

Regular Meeting, April 16, 1945

A Family with Congenitally Subluxated Lenses. DR. ROBERT R. CHACE.

This paper will be published in a later issue of the ARCHIVES.

Marfan's Syndrome with Unusual Complications: Report of a Case. DR. JAMES L. MCGRAW.

This paper was published in the August 1945 issue of the ARCHIVES, page 112.

Ocular Changes Associated with Arachnodactyly. DR. RALPH I. LLOYD, Brooklyn.

This is based on a study of 18 patients under observation over periods of three to thirteen years and some 25 other patients seen over shorter periods. Of the 18 patients, 6 have been operated on, 3 to improve vision and 3 in emergencies. Of the first 3, none was successful. In 1 patient operation was a success but the vision did not improve. Results in the second case were ruined by a hemorrhage into the vitreous and in the third, by a retinal detachment which was operated on with temporary results. Of the second group, 1 patient suffered bilateral dislocation of the lenses into the anterior chamber and the result was highly satisfactory. Another suffered a unilateral dislocation into the chamber, from which the lens was removed without difficulty. Later replacement of iris in the wound, which opened from within, posterior scleral trephine and enucleation were done. The pathologist's report was epithelial invasion of the chamber. The third patient was operated on and reoperated on for retinal detachment without success. Marked changes in position of the lens were seen in 7 cases. Two are mentioned as dislocations into the anterior chamber. Three eyes in 2 patients were dislocated into the vitreous. In 3 cases the dislocation was incomplete but sufficient to uncover the pupil, with improvement in vision in one eye of each patient. These results are discouraging, but one has no way of ascertaining the number of cases of arachnodactyly with normal eyes or satisfactory status quo.

In patients with poor vision there is progressive degeneration with complications, whereas patients with better vision may go along for years without coming into the hands of any one able to recognize the situation. Persons with this condition must be handled with care and judgment, and an effort should be made to discover the safest technic for removal of the lens.

DISCUSSION

DR. THOMAS H. JOHNSON: A patient with ectopia lentis presents an important problem. If he is a child, what is to be done? No satisfactory operation has been devised for a subluxated lens. It is known that such lenses do not absorb well after needling. If the lens is removed with a loop, there is danger of subsequent detachment of the retina, regardless of the age of the patient. In the general discussion of this paper, it would be interesting to have opinions as to the type of operation advisable and to hear what complications have occurred in the experience of others.

DR. JAMES W. SMITH: Twenty years ago Ormond and Williams noted that ectopia lentis, iridodonesis and miosis occurred in about 50 per cent of all cases of Marfan's syndrome. Interest of American ophthalmologists in these ocular changes was stimulated by the paper of Dr. Lloyd's, read before the American Ophthalmological Society in 1934.

Ectopia lentis occasionally occurs independently of arachnodactyly. Kredbova observed 37 members of a family through four generations, 15 of whom had ectopia, 2 congenital coloboma and 6 myopic changes in the fundus. None of his patients presented typical arachnodactyly. The incidence of ectopia lentis in 75 per cent of the cases of arachnodactyly and analogous anomalies of growth recently reported suggests that the etiologic factors responsible for arachnodactyly are also involved with displacement of the lens.

Dr. Lloyd, in his opening statements, properly emphasized the progressive ocular changes seen with this disorder. The small spherophakic lenses are usually dislocated upward. Later they may appear in the anterior chamber and precipitate glaucomatous symptoms. Complete dislocation into the vitreous, with loss of vision, is a common complication. Uveal degeneration and retinal detachment are two serious factors stressed by Dr. Lloyd which account for his conservative, nonsurgical approach.

Six of the 18 patients observed by Dr. Lloyd had operations. Of the 3 patients for whom operation was optional, 1 had an uncomplicated course but no visual improvement. The second patient had a hemorrhage into the vitreous. The patient referred to me for iridectomy and extraction of the lens of the left eye made an uneventful recovery, with vision limited to 20/60 + 2. Retinal separation occurred thirteen months later, the detached portion being partly replaced by discleral coagulation. Vision at present, two years later, is 20/80. In 1 of the 3 remaining patients, the lens was removed from the anterior chamber of both eyes for bilateral dislocation, but vision failed nine years later as a result of a pathologic process of the uveal tract. The eye of a second patient was enucleated after removal of the lens from the anterior chamber, and the third patient had an operation for retinal detachment without success. I agree that as long as useful vision is obtained through the aphakic pupillary area with a correcting lens, surgical intervention should not be comtemplated.

The operative failures in Dr. Lloyd's series make me hesitate to suggest a new surgical approach. I do not believe that these young, structurally defective, globes should be subjected to extraction of the lens with unavoidable loss of vitreous. Needling of the elusive subluxated lens is difficult and inadequate and must be repeated.

Several years ago Dr. Arnold Knapp called attention to Bowman's technic, in which two knife needles were used. The patient presented in the adjoining room was first examined in 1934. He was 20 years old and had worn glasses for fifteen years. Minus 15.00 D. spheres corrected vision in the right eye to 10/200 and in the left eye to 15/100 ∓. The maximum acuity obtained in the right eye with a — 30.00 D. sphere was 15/100 and that in the left eye with a —21.00 D. sphere was 15/50. No gross pathologic process was seen in the right fundus, and the macula was not involved. The lens was luxated nasally. The anterior chamber was shallow, and only moderate iridodonesis was present. The left anterior chamber was deep, and tremor of the iris was pronounced. The temporal equator was displaced posteriorly but remained attached nasally. No details of the fundus were discernible through the small aphakic area.

Three months later the left lens had wandered into the anterior chamber. Needling, attempted with the patient lying face down, failed. Later, with the pupil contracted and the patient in the sitting position, a knife needle was placed in the cornea 3 mm. from the temporal limbus; the posterior capsule was engaged, and the lens was forced against the cornea. A generous section was made in the posterior capsule, and cortical clouding began in two days. Two weeks later most of the loose cortex fell into the vitreous and was resorbed. The shrunken lenticular sac is still seen behind the iris inferiorly. Vision corrected with an aphakic lens is 20/40 +, and the patient, eleven years after discission, reads Jaeger type 2.

The same operation was performed on the right eye in 1940, when the patient was 26 years of age. Clearing of the lens was slower, and a capsulotomy had to be done. Vision with correction was 20/60, and the patient reads Jaeger type 5 plus.

When Dr. Lloyd referred the girl aged 14 to me in 1941, I was anxious to perform a needling of the posterior capsule, but he preferred extraction by spooning. The lens was sharply curved anteriorly and measured 8 mm. long and 6 mm. wide in the anteroposterior diameter. It weighed only 0.125 Gm., as compared with 0.158 Gm., the average weight for a lens at this age. The patient made an interesting diagnosis of further luxation in the right eye while she was in bed convalescing from my operation for retinal detachment on the left eye. She observed that the aphakic glass prescribed for the left eye after extraction of the lens one year previously afforded remarkable vision in the right eye. She had made this test many times after extraction of the lens, but without result.

I consider the technic which I use in cases of subluxated lenses ideally suited for patients up to the age of 20 years. Glaucoma does not occur as a complication, and the vitreous seems to tolerate young lens cortex. The feature which appeals to me particularly is that the globe is not opened and the vitreous is not lost.

Recent studies of the cause of retinal detachment are pertinent to the present discussion of operations on the lens in children. Dr. Knapp's paper on the operative prognosis of congenital cataract (Operative Prognosis of Congenital Cataract, ARCH. OPHTH. 32: 519 [Dec.] 1944) was a survey of 400 cases of retinal detachment. In 6, or nearly 16 per cent, of 37 cases of retinal detachment following cataract operations, congenital cataract had been present. Shapland (*Tr. Ophth. Soc. U. Kingdom* 54: 176, 1934) reviewed 651 cases of simple detachment of the retina in Moorfield's Eye Hospital; in about 10 per cent of these the patient was aphakic. An analysis of 40 eyes in which needling had been performed in infancy or early childhood for congenital cataract showed that retinal detachment developed in 82.5 per cent. The average interval was twenty-four and six-tenths years, the shortest being six years and the longest forty-one years. My purpose in citing these statistics is to stress the fact that, like ectopia lentis, congenital cataract is a form of defective structural development.

I view with misgiving the practice of subjecting patients with congenital cataract to innumerable needlings, and I should like to ask operations may contribute to, or actually hasten, Dr. Lloyd whether he does not feel that repeated

the appearance of retinal separation. It is my own practice to perform a generous discission of the anterior capsule in young children. If the angle of the chamber should become obstructed, the loose cortex can be irrigated out of the anterior chamber through a small keratome section. In order to avoid subjecting such eyes to repeated trauma, I have on occasion continued the needling through the posterior capsule. As in cases of ectopia lentis, the posterior discission lessens the possibility of glaucoma developing postoperatively. I should like to ask Dr. Lloyd whether he has any objection to opening the posterior capsule. Will he tell us whether ectopia lentis is found only in cases of Marfan's syndrome?

The Section is indebted to Dr. Lloyd for popularizing a syndrome considered originally to be of interest only to pediatricians and orthopedists. He has directed our attention to the fact that the ocular complications are very common. His youngest patient was 6 years of age. If ophthalmologists follow his suggestion of dilating the pupils in very young children, they may be in a position occasionally to establish the diagnosis before serious skeletal defects are evident clinically.

DR. HERMANN BURIAN, Hanover, N. H. (by invitation): The statement on the program that I am going to discuss Dr. Lloyd's paper is somewhat of a misrepresentation. Even though I have for a long time been interested in the syndrome of arachnodactyly with subluxation of the crystalline lens, I should not presume to discuss Dr. Lloyd's masterly presentation.

I am here only because Dr. Lloyd, when in Hanover last summer, saw a case of arachnodactyly in which I had operated, and he asked me to tell you about it. My comments will be restricted to the report of this case.

R. J. L., a white boy born Dec. 12, 1936, was first seen by me on May 19, 1942, when he was 5½ years of age. There was no relevant family history of ocular disease. The 3 siblings had normal eyes.

The patient had never been seriously ill. He had developed normally and, according to the mother, had shown no signs of mental retardation. However, a diagnosis of congenital heart murmur had been made.

The child had always held objects close to his eyes and had exhibited other signs of poor vision; these had become particularly noticeable in the year prior to examination.

Examination revealed typical signs of arachnodactyly. His arms and legs, particularly his fingers and toes, were very long and thin. The eyes were white and quiet. The irises showed a normal structure but exhibited pronounced iridodonesis. "Spider-web" remnants of the pupillary membrane were seen in the upper part of both pupils, particularly in the right eye. Both crystalline lenses were subluxated downward and nasally, so that the upper edge of the lens appeared in the lower part of the undilated pupil. The fundi were somewhat depigmented but presented no anomaly. Uncorrected visual acuity was less than 20/400 in each eye; vision could not to be improved with glasses. It was difficult, however, to make subjective tests on the rather unresponsive patient. A diagnosis of Marfan's syndrome was made, and hospitalization was advised.

In June 1942 the patient was admitted to the hospital for examination and treatment. Physical examination revealed no noteworthy anomaly aside from the arachnodactyly. The heart was normal except for a rapid rate. There was reduplication of the second sound at the apex, which was heard better medially. The pulsations of the dorsalis pedis artery were weak, but those of the popliteal artery were normal. The Vollmer patch test for tuberculosis gave a negative response. Complete blood counts, serologic tests and urinalysis gave normal results. Roentgenograms of the chest, skull and upper extremities revealed nothing abnormal. An electrocardiogram was essentially normal.

On June 25 a discission of the right lens was attempted. Two discission knives were introduced into the anterior chamber, one from each side, at 4 and 8 o'clock respectively. The lens was held with one knife and the discission performed with the other. No clouding of the lens was obtained, and on June 30 extraction of the right lens through a keratome incision was attempted. Grasping of the lens with the capsule forceps did not succeed, and the lens was delivered by a loop, with considerable loss of vitreous. The condition of the eye quieted down, but the pupil was distorted upward; and on October 1 an iridotomy was performed by introducing a discission knife into the anterior chamber near the limbus at 12 o clock. A section through the iris was made in the vertical meridian, and a good opening in the iris was obtained.

The boy was readmitted in January 1943. He had a slight elevation of temperature, and operation was postponed for a few days. There were enlarged anterior cervical glands. One observer thought that there was a soft, late systolic murmur at the apex, and another observer heard a diastolic murmur at the apex. But the next day, with a slower heart rate, this resolved itself into a reduplication of the heart sounds, as noted on the first admission. For two days shortly after his operation he again had a rather high fever, and it was thought at the time that he had grip, but the lungs were clear and there were only a cough, enlarged cervical glands and reddening of the throat. Blood counts and urinalysis again gave normal results.

On Feb. 5, 1943 the left lens was removed. The anterior chamber was opened with a keratome at the upper limbus. Pressure was exerted on the lower limbus and the lens grasped with

intracapsular forceps and lifted into the anterior chamber. It was removed from the chamber by combined pressure and traction. The lens proved to be small and globular. No vitreous was lost.

In spite of the smoothness of the operative procedure and the application of physostigmine, the upper part of the iris became inverted, and the pupil, accordingly, was distorted to such an extent that it was covered by the upper lid.

On July 6, 1943 an iridotomy was performed on the left eye. The discission knife was inserted through the upper limbus and the iris sectioned in the vertical meridian with sawing movements. The postoperative course was uneventful.

The patient was followed through 1943 and 1944; when he was last seen, on Nov. 13, 1944, the condition was as follows: Both eyes were white and quiet. The pupils were triangular, the right one being smaller than the left. The pupillary areas were black, and the fundi appeared normal. The best vision was obtained with a correction of + 10.00 D. sph. ⊃ + 2.00 D. cyl., axis 180, which gave visual acuity of 20/60 + 2 in the right eye, 20/60 in the left eye and 20/50 + binocularly. The keratometric readings showed astigmatism of — 6.50 D. at axis 93 in the right eye and — 6.00 D. at axis 100 or 110 in the left eye.

Dr. Clyde E. McDannald: May I ask Dr. Lloyd whether there are any statistics relating to hereditary transmission? I have not heard of any.

Dr. Willis S. Knighton: I should like to report the case of a man 27 years of age in which there were features which differed a little from those reported tonight. The patient was well built but had all the characteristics of arachnodactyly. The interesting observation on refraction was that the lenses were dislocated downward, so that the upper border was exactly in the center of the pupil in each eye, and his vision could be improved by correction with either plus or minus spheres. He was very uncomfortable with minus spheres, and vision was only 20/70 in each eye; but with plus spheres vision was 20/40 in the right eye and 20/20 in the left eye. My colleagues and I had planned to do an intracapsular extraction with iridectomy, but the response of the pupil to the mydriatic was so great that the iris was just a narrow band; the lens was therefore removed through a round pupil. As soon as the section was made, the lens floated up toward the cornea and was delivered with a loop alone; almost no traction was necessary, and no counterpressure was made. There was no loss or presentation of vitreous. Convalescence was uncomplicated, and his vision was 20/20— with correction. The astonishing thing was that with plus spheres for his right, completely aphakic, eye and for his left, half aphakic, eye he had comfortable binocular vision. Dr. Lloyd suggested that we remove the lens of the left eye. The patient did not return to the clinic for two years; although he had been doing hard manual labor, there was no further dislocation of the lens in the left eye. Extraction of the lens in the left eye was performed, with the same result. He was last seen four years after the first operation and two years after the second operation. At that time he had 20/20— vision in each eye, with good binocular vision.

Dr. Ralph I. Lloyd, Brooklyn: Certainly, all luxations of the lens are not due to arachnodactyly, but all congenital bilateral luxations should be considered as associated with arachnodactyly unless proved otherwise. When a case of bilateral luxation is found, the family tree should be thoroughly studied to find a transmitter of this, usually dominant, trait. The parent may have normal bony structure and mild luxation with good vision, or he may have normal eyes and the characteristic changes of the bones. Persons with either type may have offspring with typical arachnodactyly. When operation to improve vision must be done, younger patients seem to fare better than adolescents and adolescents better than adults. As a rule the lens can be easily removed without using the spoon, for such a lens is lighter and smaller than the normal lens. It will float up into the wound if an iridectomy is done. The best operation is the one that disturbs the eye the least, and probably needling is safer. I have seen Dr. Smith do the operation he has described here; although it is slow and may have to be done again, the results are very good. However, I am not committed absolutely to any one procedure, and I prefer to play out the hand as the cards may fall. It is plain from what has been said that cases of this syndrome are not a favorable group for any operation.

Traumatic Staining of the Conjunctiva with Indelible Lead: Report of a Case. Dr. Harold Wass.

This case is presented because of the bizarre clinical appearance and because of the excellent result obtained with subconjunctival irrigation with saline solution. The patient was seen in the emergency clinic of the New York Eye and Ear Infirmary, in the service of Dr. Willis Knighton, one-half hour after the conjunctiva was lacerated with the point of an indelible pencil. Pieces were removed from under the conjunctiva with forceps, and the cul-de-sac was irrigated freely with warm saline solution. Many pieces and grains of lead remained under the conjunctiva, and the whole globe was stained a brilliant violet.

Irrigation of the subconjunctival space with saline solution and closure of the conjunctival wound with black silk sutures were done in the surgical service; this treatment resulted in complete fading of the stain in forty-eight hours.

In addition to the mechanical cleansing of the subconjunctival space, the black silk sutures may have stimulated phagocytosis by acting as an additional foreign body. However, the same indelible lead was inserted below the conjunctiva in 1 rabbit eye and into the vitreous cavity of another. Enucleation was performed two weeks later, and microscopic examination of the slides showed no cellular reaction in either globe and nothing resembling phagocytosis. The indelible lead remained relatively inert in the rabbit eye.

The sections of the rabbit eyes were prepared by Dr. E. B. Burchell, of the Eno Laboratory of the New York Eye and Ear Infirmary, and the Kodachromes were made by the department of photography. None of the bleaches used by the personnel of the manufacturing companies are suitable for ophthalmic use.

DISCUSSION

DR. WILLIS S. KNIGHTON: A point of practical importance is the fact, according to Dr. Sitchevska, that self-inflicted wounds of this sort were rather common in the Russian army. In view of the clinical course in this case, however, the method does not seem a very practical means of escaping military duty, because the dye disappears so rapidly. However, the picture is so striking at first, when the color is present, that I can see how a draft board, or similar body, would give a man temporary leave at least.

Response of Chronic Simple Glaucoma to Treatment with Cyclodiathermy Puncture.

DR. FREDERICK W. STOCKER, Durham, N. C.

This paper was published in full in the September issue of the ARCHIVES, page 181.

DISCUSSION

DR. ARNOLD KNAPP: Dr. Stocker has undoubtedly covered this point in his admirable presentation, but I should like the ask him to tell us again whether he has ever seen any damage done to an eye by this operation. It has seemed to me that it is difficult to gage just the number of diathermy punctures required in a particular case and that it is easy to do too much.

The statement that notwithstanding atrophy of a part of the ciliary body eyes with this condition do not show any subsequent deterioration is particularly interesting.

I should like to ask Dr. Stocker whether he has tried this operation in a case of secondary glaucoma following thrombosis of the central retinal vein.

DR. G. BONACCOLTO: Years ago I was interested in Dr. Lagrange's work on *colmatage* in cases of detachment of the retina. This consisted in electrocauterization of the sclera, first at the inferior and, a few weeks later, at the superior limbal region. He had observed intraocular hypotension in eyes with detachment of the retina. With this procedure he produced intraocular hypertension, which, in his opinion, should help to push the retina into place.

I performed this operation in a few cases of detachment of the retina during the period from 1928 to 1932. I obtained moderate hypertension, which lasted as long as I was able to follow the patients.

Will Dr. Stocker explain the reason for the difference in the results obtained by him and by Lagrange when they used similar procedures?

DR. KAUFMAN SCHLIVEK: It is a rare opportunity to listen to a paper describing the results of a new operation covering 100 cases or more. Many years ago I performed this operation in a number of cases of absolute glaucoma, and in the past two and a half years I have used the same method in 4 cases of glaucoma with vision; in all instances the results were satisfactory. One patient was a Negro woman who had had several operations for glaucoma on her left eye, and the eye was blind. The other eye had chronic simple glaucoma with good vision and rather poor fields; the operation recommended by Dr. Stocker was done, and the result was excellent. The other 3 patients had had several operations, without benefit; this operation controlled the tension, and vision was retained.

DR. WILLIS S. KNIGHTON: As I understand it, glaucoma can be caused either by obstruction to the outflow of the aqueous or by an increase in the inflow of the aqueous. This operation is designed apparently to reduce the inflow of the aqueous. I should like to ask Dr. Stocker whether he would try to reduce the production of aqueous almost to the vanishing point in cases in which the obstruction to the outflow is nearly complete.

DR. FREDERICK W. STOCKER, Durham, N. C.: To answer Dr. Knapp first: One must keep distinct experience in cases of chronic simple glaucoma and experience in cases of secondary glaucoma in which this operation has already been performed a number of times or in which two or three other operations have been done without success. I used to employ considerably stronger current and larger needles in these cases of long standing, and I believe that with this technic I should not have dared to approach the good eyes with chronic simple glaucoma. But by using a very short needle and very little current, as I did in this series of cases of chronic glaucoma in which no other operation had been employed, I have not observed any permanent damage to the eye. I have called this report a preliminary one because I may have to watch the patients for a longer time. However, I think that if after eight or ten months one cannot see any appreciable damage to the eye, one is justified in doing more operations of this kind.

Glaucoma associated with thrombosis of the central retinal vein is in another category; and I have treated several patients with this type.

In one such patient the eye was cured of its glaucoma for almost two years after the first thrombosis occurred and was painless, and the vitreous had cleared up to such a degree that I could see the strange fundus picture of healed thrombosis of the central vein. So the eye had completely recovered, but two years later the patient had a tremendous hemorrhage in the vitreous, which led to enucleation. I think one is justified in using this method in cases of thrombosis of the retinal vein causing glaucoma, for I do not know of any other method which will give such good results. I believe that with a secondary type of glaucoma cyclodiathermy puncture is of great value, and chronic inflammatory and painful eyes have a remarkable tendency to become painless. Even if the tension is not quite reduced to normal, the patient does not suffer any more.

The hypotension associated with detached retina, so far as I could understand from Dr. Bonaccolto, was treated with the application of electrocautery, which is a different type of treatment than cyclodiathermy puncture. With the diathermy cautery one does not produce so much inflammation on the surface of the sclera, as it is at the point of the needle that the most intensive coagulating action is to be expected. I cannot explain offhand why the electrocautery should produce permanent hypertension. As I said, my associates and I observed temporary hypertension for several days in a few cases; but, as I also demonstrated, this does not mean anything in the final outcome, for those cases in which there was a relatively high tension after the operation were not among those in which tension was high at the end.

I am encouraged to hear from Dr. Schlivek that in 1 case of simple chronic glaucoma in which no treatment had previously been employed this operation had given satisfactory results in the hands of some one else.

In answer to Dr. Knight's question about the theory of inflow and outflow of aqueous, I had to limit myself to the simplest principles in order to demonstrate what is involved if one wishes to reduce the tension by achieving a decrease in inflow of liquid. In cases of simple chronic glaucoma, with a tension of about 30 or 35 mm. of mercury, one would not expect complete blockage of the outflow. I do not know how much of the ciliary body would have to be destroyed when complete obstruction is present. In cases of secondary glaucoma, for instance, with thrombosis of the central vein, in which the iris is pushed forward and is adherent to the posterior surface of the cornea, one might have almost complete blockage of the angle of the chamber. But it is known that there are other possible paths of outflow in the eye. The iris and the choroid can absorb fluid, and there is a flow of fluid through the vitreous along the canal of Cloquet. Therefore it is almost impossible to determine when the outflow would be 100 per cent blocked.

Book Reviews

The Psychology of Seeing. By Herman F. Brandt, Ph.D. Price $3.75. Pp. 240. New York: Philosophical Library, Inc., 1945.

This small volume is misnamed, because it is not a consideration of the psychology of seeing but a study of the results of behavior patterns obtained by means of ocular photography.

The author has invented a camera for recording eye movements. In many ways it is similar to the score or more of eye movement cameras which preceded it and which culminated in the ophthalmograph with which ophthalmologists are familiar. It has the advantage that the movements of the eyes in observing a double page magazine layout (such as *Saturday Evening Post* or *Life*) can be photographed on a single stationary film and the progress of the eye movements studied from a projection of the photograph back on to the original copy.

Of the eight parts of the book, the first is an elementary discussion of psychology, the second a description of the camera and the third a demonstration of basic eye movements. The fourth part is devoted to advertising as evaluated by means of the author's camera. Part V is the most scientific chapter of the book, in which various problems of learning are studied by means of photographs of eye movements. It is largely from this chapter that the ophthalmologist would derive any profit. Part VI is a discussion of art as judged by camera studies of eye movements; part VII discusses factors involved in ocular patterns, and in the final chapter the author projects future studies to be carried out, using his instrument and technics, in such diverse fields as crime detection and talking books.

The book is largely written for the advertising industrialist, who wants to get his message, pictorial or printed, in a preferred position and to grasp and hold the attention of readers against the clamor of competitors. It has little to do with ophthalmology except for the parts relating to reading and learning difficulties. These are discussed diagnostically, with few if any therapeutic suggestions. LæGrand H. Hardy.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 17 Horseferry Rd., London, England.

Pan-American Congress of Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretaries: Dr. Conrad Berens, 35 E. 70th St., New York. Dr. M. E. Alvaro, 1511 Rua Consolacão, São Paulo, Brazil.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chengtu Ophthalmological Society

President: Dr. Eugene Chan.
Secretary: Dr. K. S. Sun.
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, China.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

Faculty of Ophthalmologists

President: Brig. Sir Stewart Duke-Elder, 63 Harley St., London, W. 1, England.
Secretary: Mr. Frank W. Law, 45 Lincoln's Inn Fields, London, W. C. 2., England.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. I. Imre, Budapest.
Assistant Secretary: Dr. Stephen de Grósz, University Eye Hospital, Máriautca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

North of England Ophthalmological Society

President: Mr. John Foster, 45 Park Sq., Leeds.
Secretary: Mr. William M. Muirhead, 70 Upper Hanover St., Sheffield.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

Ophthalmological Society of Australia

President: Dr. N. McAlister Gregg, 193 Macquarie St., Sydney.
Secretary: Dr. D. A. Williams, 27 Commonwealth St., Sydney.
Place: Sydney:. Time: Oct. 3-6, 1945.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the secetary, Dr. Mohammed Khalil.

Ophthalmological Society of Hospital de Nuestra Señora de la Luz

Chairman: Dr. Manuel J. Icaza y Dublán, México, D. F., Mexico.
Secretary: Dr. Jorge Meyrán, México, D. F., Mexico.

Ophthalmological Society of South Africa

President: Dr. A. W. Sichel, National Mutual Bldg., Church Square, Cape Town.
Secretary: Dr. J. K. de Kock, Groote Kerk Bldg., 32 Parliament St., Cape Town.

Ophthalmological Society of the United Kingdom

President: Mr. Charles B. Goulden, 89 Harley St., London.
Secretary: Mr. Frank W. Law, 30 Devonshire Pl., London, W. 1.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel. Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Mr. P. G. Doyne, 60 Queen Anne St., London, W. 1, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Col. F. A. Juler, 96 Harley St., London, W. 1, England.
Secretary: Dr. Harold Ridley, 60 Queen Anne St., London, W. 1, England.

SÃO PAULO SOCIETY OF OPHTHALMOLOGY

President: Silvio de Almeida Toledo, Barão de Ilapetininga St., 88, 5º Andar, São Paulo, Brazil.
Secretary: Dr. Plinio de Toledo Piza, Enfermaria Santo Luzia, Santa Casa de Misericordia, Cesario Motta, St. 112, São Paulo, Brazil.

SCOTTISH OPHTHALMOLOGICAL CLUB

President: Dr. S. Spence Meighan, 13 Woodside Pl., Glasgow, C. 3.
Secretary: Dr. Alexander Garrow, 15 Woodside Pl., Glasgow, C. 3.
Place: Edinburgh and Glasgow, in rotation.

SOCIEDAD ARGENTINA DE OFTALMOLOGIA

Chairman: Dr. Jorge Malbrán, Buenos Aires.
Secretary: Dr. Benito Just Tiscornia, Santa Fe 1171, Buenos Aires.

SOCIEDAD OFTALMOLOGIA DEL LITORAL, ROSARIO (ARGENTINA)

President: Prof. Dr. Carlos Weskamp, Laprida 1159, Rosario.
Secretary: Dr. Arturo Etchemendigaray, Villa Constitución, Santa Fé.
Place: Rosario. Time: Last Saturday of every month, April to November. All correspondence should be addressed to the President.

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAIS

President: Prof. Hilton Rocha, Rua Rio de Janeiro 2251, Bello Horizonte, Minas Geraes, Brazil.
Secretary: Dr. Ennio Coscarelli, Rua Aimorés 1697, Bello Horizonte, Minas Geraes, Brazil.

SOCIEDADE DE OFTALMOLOGIA E OTORRINOLARINGOLOGIA DE RIO GRANDE DO SUL

President: Dr. Luiz Assumpção Osorio, Edificio Vera Cruz, Apartamento 134, Porto Alegre, Rio Grande do Sul.
Secretary: Dr. Fernando Voges Alves, Caixa Postal 928, Porto Alegre, Rio Grande do Sul.

SOCIEDADE DE OPHTHALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4, III tr., Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Secretary: Dr. R. J. Masters, 23 E. Ohio St., Indianapolis.
In compliance with the request of the Office of Defense Transportation and in the interest of the national war effort a meeting will not be held in 1945.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
President-Elect Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary-Treasurer: Dr. Walter S. Atkinson, 129 Clinton St., Watertown, N. Y.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. Conrad Berens, 35 E. 70th St., New York.
Secretary-Treasurer: Major Brittain F. Payne, School of Aviation Medicine, Randolph Field, Texas.
Assistant Secretary-Treasurer: Dr. Hunter Romaine, 35 E. 70th St., New York.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

Canadian Ophthalmological Society

President: Walter W. Wright, 170 St. George St., Toronto 5.
Secretary-Treasurer: Dr. Kenneth B. Johnston, Suite 1, 1509 Sherbrooke St. W., Montreal.

National Society for the Prevention of Blindness

President: Mr. Mason H. Bigelow, 1790 Broadway, New York.
Secretary: Miss Regina E. Schneider, 1790 Broadway, New York.
Executive Director: Mrs. Eleanor Brown Merrill, 1790 Broadway, New York.

SECTIONAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear Nose and Throat

President: Dr. N. Zwaifler, 46 Wilbur Ave., Newark.
Secretary: Dr. William F. Keim Jr., 25 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Central Illinois Society of Ophthalmology and Otolaryngology

President: Dr. Watson Gailey, 1000 N. Main St., Bloomington, Ill.
Secretary-Treasurer: Dr. William F. Hubble, 861-867 Citizens Bldg., Decatur, Ill.

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

New England Ophthalmological Society

President: Dr. Theodore L. Terry, 140 Marlborough St., Boston.
Secretary-Treasurer: Dr. Merrill J. King, 264 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. D. H. O'Rourke, 1612 Tremont Pl., Denver.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. James H. Mathews, 1317 Marion St., Seattle, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 301 Stimson Bldg., Seattle 1.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month except June, July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. J. Sheldon Clark, 27 E. Stephenson St., Freeport, Ill.
Secretary-Treasurer: Dr. Harry R. Warner, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. L. D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. Harold H. Heuser, 207 Davidson Bldg., Bay City, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July, August and September.

Sioux Valley Eye and Ear Academy

President: Dr. J. C. Decker, 515 Francis Bldg., Sioux City, Iowa.
Secretary-Treasurer Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. John H. Burleson, 414 Navarro St., San Antonio, Texas.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville, S. C.

Southwestern Academy of Eye, Ear, Nose and Throat

President: Dr. H. L. Brehmer, 221 W. Central Ave., Albuquerque, N. Mex.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.

Southwestern Michigan Triological Society

President: Dr. W. M. Dodge, 716 First National Bank Bldg., Battle Creek.
Secretary-Treasurer: Dr. Kenneth Lowe, 25 W. Michigan Ave., Battle Creek.
Time: Last Thursday of September, October, November, March, April and May.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. Ray Parker, 218 Franklin St., Johnstown, Pa.
Secretary-Treasurer: Dr. J. McClure, Tyson, Deposit Nat'l Bank Bldg., Dubois.

STATE

Arkansas State Medical Society, Eye, Ear, Nose and Throat Section

President: Dr. Raymond C. Cook, 701 Main St., Little Rock.
Secretary: Dr. K. W. Cosgrove, Urquhart Bldg., Little Rock.

Colorado Ophthalmological Society

President: Dr. C. A. Ringle, 912–9th Ave., Greeley.
Secretary: Dr. W. A. Ohmart, 1102 Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. F. L. Phillips, 405 Temple St., New Haven.
Secretary-Treasurer: Dr. W. H. Turnley, 1 Atlantic St., Stamford, Conn.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. William O. Martin Jr., Doctors Bldg., Atlanta.
Secretary-Treasurer: Dr. C. K. McLaughlin, 526 Walton St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. McK. Ruby, Union City.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. K. Von Lackum, 117–3d St. S. E., Cedar Rapids.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

Kansas State Medical Society, Section on Ophthalmology and Otolaryngology

President: Dr. W. D. Pittman, Pratt.
Secretary: Dr. Louis R. Haas, 902 N. Broadway, Pittsburg.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Val. H. Fuchs, 200 Carondelet St., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Medical Society of the State of Pennsylvania, Section on Eye, Ear, Nose and Throat Diseases

Chairman: Dr. Karl M. Houser, 2035 Delancey St., Philadelphia 3.
Secretary: Dr. William T. Hunt Jr., 1205 Spruce St., Philadelphia 7.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Edmond L. Cooper, 1553 Woodward Ave., Detroit 26.
Secretary: Dr. Ralph H. Gilbert, 110 Fulton St. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Karl C. Wold, 1051 Lowry Bldg., St. Paul 2.
Secretary: Dr. William A. Kennedy, 372 St. Peter St., St. Paul 2.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. William Morrison, 208 N. Broadway, Billings.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

Nebraska Academy of Ophthalmology and Otolaryngology

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N. St., Lincoln.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. George P. Meyer, 410 Haddon Ave., Camden.
Secretary: Dr. John P. Brennan, 429 Cooper St., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Harold J. Joy, 504 State Tower Bldg., Syracuse 2.
Secretary: Dr. Maxwell D. Ryan, 660 Madison Ave., New York 21.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. Hugh C. Wolfe, 102 N. Elm St., Greensboro.
Secretary: Dr. Vanderbilt F. Couch, 104 W. 4th St., Winston-Salem.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. L. Diven, City National Bank Bldg., Bismarck.
Secretary-Treasurer: Dr. A. E. Spear, 20 W. Villard, Dickenson.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul Neely, 1020 S. W. Taylor St., Portland.
Secretary-Treasurer: Dr. Lewis Jordon, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Pennsylvania Academy of Ophthalmology and Otolaryngology

President: Dr. Lewis T. Buckman, 83 S. Franklin St., Wilkes-Barre.
Secretary Pro Tem: Dr. Paul C. Craig, 232 N. 5th St., Reading.
Time: Last week in April.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society, Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. J. L. Sanders, 222 N. Main St., Greenville.
Secretary: Dr. J. H. Stokes, 125 W. Cheves St., Florence.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. F. H. Rosebrough, 603 Navarro St., San Antonio.
Secretary: Dr. M. K. McCullough, 1717 Pacific Ave., Dallas.

Utah Ophthalmological Society

President: Dr. E. B. Fairbanks, 315 Medical Arts Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Dean Spear, 516 Boston Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Mortimer H. Williams, 30½ Franklin Rd. S. W., Roanoke.
Secretary-Treasurer: Dr. Meade Edmunds, 34 Franklin St., Petersburg.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. E. L. Mather, 39 S. Main St., Akron, Ohio.
Secretary-Treasurer: Dr. V. C. Malloy, 2d National Bank Bldg., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. B. M. Cline, 153 Peachtree St. N. E., Atlanta, Ga.
Acting Secretary: Dr. A. V. Hallum, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., fourth Monday of each month, from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. Luther E. Wilson, 919 Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn 11.
Secretary-Treasurer: Dr. Louis Freimark, 256 Rochester Ave., Brooklyn 13.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. William H. Howard, 389 Linwood Ave., Buffalo 9.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo 9.
Time: Second Thursday of each month from October to May.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Samuel J. Meyer, 58 E. Washington St., Chicago 2.
Secretary: Dr. W. A. Mann, 30 N. Michigan Ave., Chicago 2.
Place: Continental Hotel, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati General Hospital Ophthalmology Staff

Chairman: Dr. D. T. Vail, 441 Vine St., Cincinnati.
Secretary: Dr. A. A. Levin, 441 Vine St., Cincinnati.
Place: Cincinnati General Hospital. Time: 7:45 p. m., third Friday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. M. Paul Motto, Rose Bldg., Cleveland.
Secretary: Dr. H. H. Wygand, Guardian Bldg., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Clerk: Dr. George F. J. Kelly, 37 S. 20th St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Erwin W. Troutman, 21 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. T. Rees Williams, 380 E. Town St., Columbus 15, Ohio.
Place: University Club. Time: 6:15 p. m., first Monday of each month, from October to May, inclusive.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. C. B. Collins, 704 Medical Professional Bldg., Corpus Christi, Texas.
Secretary: Dr. L. W. O. Janssen, 710 Medical Professional Bldg., Corpus Christi, Texas.
Time: 6:30 p. m., third Tuesday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Ruby K. Daniel, Medical Arts Bldg., Dallas 1, Texas.
Secretary: Dr. Tom Barr, Medical Arts Bldg., Dallas 1, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Wesley G. Reid, 667 Fisher Bldg., Detroit 2.
Place: Club rooms of Wayne County Medical Society.
Time: First Monday of each month, November to April, inclusive.

Detroit Ophthalmological Society

President: Dr. Raymond S. Goux, 545 David Whitney Bldg., Detroit 26.
Secretary: Dr. Arthur Hale, 1609 Eaton Tower, Detroit 26.
Place: Club rooms of Wayne County Medical Society. Time: 6:30 p. m., third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Appointed at each meeting.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Rex Howard, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Gough, Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. Lyle J. Logue, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. John T. Stough, 803 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmoological and Oto-laryngological Society

President: Dr. Myron Harding, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Edgar Johnson, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Francis Carl Hertzog, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Robert G. Thornburgh, 117 E. 8th St., Long Beach, Calif.
Place: Seaside Hospital. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Secretary-Treasurer: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach 2, Calif.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Lower Anthracite Eye, Ear, Nose and Throat Society

Chairman: Each member in alphabetical order.
Secretary: Dr. James J. Monohan, 31 S. Jardin St., Shenandoah, Pa.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.
Secretary: Dr. Frazier Williams, 1801 I St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. Frank G. Treskow, 411 E. Mason St., Milwaukee 2.
Place: University Club. Time: 6:30 p. m., fourth Tuesday of each month from October to May.

Montreal Ophthalmological Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6.30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.
Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. M. M. Cullom, 700 Church St., Nashville, Tenn.
Secretary: Dr. R. E. Sullivan, 432 Doctors Bldg., Nashville, Tenn.
Place: James Robertson Hotel. Time 6:30 p. m., third Monday of each month from October to May.

New Haven Ophthalmological Society

President: Dr. William H. Ryder, 185 Church St., New Haven, Conn.
Secretary: Dr. Frederick A. Wiess, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Oto-laryngological Society

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Secretary: Dr. Truman L. Boyes, 654 Madison Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Maurice L. Wieselthier, 1322 Union St., Brooklyn.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York 28.
Place: New York Academy of Medicine, 2 E. 103d St. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Oklahoma City Academy of Ophthalmology and Otolaryngology

President: Dr. James P. Luton, 117 N. Broadway, Oklahoma City.
Secretary: Dr. Harvey O. Randel, 117 N. Broadway, Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. A. A. Steinberg, 1502 Farnam St., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha 2.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m. dinner; 7 p. m. program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. Thomas Sanfacon, 340 Park Ave., Paterson, N. J.
Secretary-Treasurer: Dr. J. Averbach, 435 Clinton Ave., Clinton, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

President: Dr. Isaac Tassman, 136 S. 16th St., Philadelphia.
Secretary: Dr. Glen Gregory Gibson, 255 S. 17th St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. George H. Shuman, 351-5th Ave., Pittsburgh.
Secretary: Dr. Robert J. Billings, 509 Liberty Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Reading Eye, Ear, Nose and Throat Society

President: Dr. Isaac B. High, 326 N. 5th St., Reading, Pa.
Secretary: Dr. Paul C. Craig, 232 N. 5th St., Reading Pa.
Place: Wyomissing Club. Time: 6:30 p. m., third Wednesday of each month from September to July.

Richmond Eye, Ear, Nose and Throat Society

President: Dr. Luther C. Brawner, Professional Bldg., Richmond, Va.
Secretary: Dr. Clifford A. Folkes, Professional Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.

St. Louis Ophthalmic Society

President: Dr. Vincent Jones, 634 N. Grand Blvd., St. Louis.
Secretary: Dr. T. E. Sanders, 508 N. Grand Blvd., St. Louis 3.
Place: Oscar Johnson Institute. Time: Fourth Friday of each month from October to April, inclusive, except December, at 8:00 p. m.

San Antonio Ophthalmo-Oto-Laryngological Society

President: Dr. Belvin Pritchett, 705 E. Houston St., San Antonio 5, Texas.
Secretary-Treasurer: Lt. Col. John L. Matthews, AAF School of Aviation Medicine, Randolph Field, Texas.
Place: San Antonio, Brooke General Hospital, Randolph Field or San Antonio Aviation Cadet Center. Time: 7 p. m., second Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Roy H. Parkinson, 870 Market St., San Francisco.
Secretary: Dr. A. G. Rawlins, 384 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Clarence A. Veasey Sr., 421 W. Riverside Ave., Spokane, Wash.
Secretary: Dr. Clarence A. Veasey, 421 W. Riverside Ave., Spokane, Wash.
Place: Spokane Medical Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. A. H. Rubenstein, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. H. Blaisdell, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. L. C. Ravin, 316 Michigan St., Toledo 2, Ohio.
Secretary: Dr. W. W. Randolph, 1838 Parkwood Ave., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. Harold M. Downey, 1740 M St. N. W., Washington, D. C.
Secretary-Treasurer: Dr. Richard W. Wilkinson, 1408 L St. N. W., Washington, D. C.
Place: Medical Society of District of Columbia Bldg., 1718 M St. N. W., Washington, D. C. Time: 7:30 p. m., first Monday in November, January, March and May.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.